In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS

Filed: July 13, 2023

DONNA OSSO,	*	PUBLISHED
Petitioner,	*	No. 18-575V
v.	*	Special Master Nora Beth Dorsey
SECRETARY OF HEALTH AND HUMAN SERVICES,	*	Entitlement; Hepatitis B Vaccine; Guillain-Barré Syndrome ("GBS").
Respondent.	*	

Brian L. Cinelli, Schiffmacher Cinelli Adoff LLP, Buffalo, NY, for Petitioner.
Kyle Edward Pozza, U.S. Department of Justice, Washington, DC, for Respondent.

RULING ON ENTITLEMENT[1]

I. INTRODUCTION

On April 20, 2018, Donna Osso ("Petitioner") filed a petition for compensation under the National Vaccine Injury Compensation Program ("Vaccine Act" or "the Program"), 42 U.S.C. § 300aa-10 et seq. (2018).[2] Petitioner alleges that she suffered Guillain-Barré Syndrome ("GBS") as the result of a hepatitis B vaccination administered on April 23, 2015. Petition at Preamble

[1] Because this Ruling contains a reasoned explanation for the action in this case, the undersigned is required to post it on the United States Court of Federal Claims' website and/or at https://www.govinfo.gov/app/collection/uscourts/national/cofc in accordance with the E-Government Act of 2002. 44 U.S.C. § 3501 note (2018) (Federal Management and Promotion of Electronic Government Services). **This means the Ruling will be available to anyone with access to the Internet.** In accordance with Vaccine Rule 18(b), Petitioner has 14 days to identify and move to redact medical or other information, the disclosure of which would constitute an unwarranted invasion of privacy. If, upon review, the undersigned agrees that the identified material fits within this definition, the undersigned will redact such material from public access.

[2] The National Vaccine Injury Compensation Program is set forth in Part 2 of the National Childhood Vaccine Injury Act of 1986, Pub. L. No. 99-660, 100 Stat. 3755, codified as amended, 42 U.S.C. §§ 300aa-10 to -34 (2018). All citations in this Ruling to individual sections of the Vaccine Act are to 42 U.S.C. § 300aa.

(ECF No. 1). Respondent argued against compensation, stating that "this case is not appropriate for compensation under the [Vaccine] Act." Respondent's Report ("Resp. Rept.") at 2 (ECF No. 14).

After carefully analyzing and weighing the evidence presented in this case in accordance with the applicable legal standards, the undersigned finds that Petitioner has provided preponderant evidence that her hepatitis B vaccine caused her GBS, satisfying Petitioner's burden of proof under Althen v. Secretary of Health & Human Services, 418 F.3d 1274, 1280 (Fed. Cir. 2005). Accordingly, Petitioner is entitled to compensation.

II. ISSUES TO BE DECIDED

Diagnosis is not in dispute. Joint Pre-hearing Submission, filed July 7, 2022, at 1 (ECF No. 83). "The parties primarily dispute (1) whether the hepatitis B vaccination can cause GBS; (2) whether [P]etitioner's GBS was caused by her receipt of a hepatitis B vaccination on April 23, 2015; and (3) overall, whether [P]etitioner has satisfied the Althen prongs." Id. at 2.

III. BACKGROUND

A. Procedural History

On April 20, 2018, Petitioner filed her petition, medical records, medical literature, and affidavits. Petition; Petitioner's Exhibits ("Pet. Exs.") 1-17. Respondent filed his Rule 4(c) Report on April 29, 2019, arguing against compensation. Resp. Rept. at 2. Petitioner filed an expert report from Dr. Marcel Kinsbourne on August 26, 2019. Pet. Ex. 18.

This case was reassigned to the undersigned on October 3, 2019. Notice of Reassignment dated Oct. 3, 2019 (ECF No. 19). Thereafter, the parties entertained settlement discussions until January 2020. Joint Status Rept., filed Jan. 15, 2020 (ECF No. 26).

Respondent filed expert reports from Dr. Lawrence Hale Moulton and Dr. Thomas Paul Leist on May 15, 2020. Resp. Exs. A, C. On October 30, 2020, Petitioner filed a responsive expert report from Dr. Kinsbourne. Pet. Ex. 19.

A Rule 5 conference was held on January 26, 2021. Rule 5 Order dated Jan. 26, 2021 (ECF No. 45). The undersigned preliminarily found Petitioner would likely be able to prove she was entitled to compensation if this case were to go to hearing. Id. at 1-2. Respondent advised he was willing to engage in informal settlement discussions. Resp. Status Rept., filed Feb. 25, 2021 (ECF No. 46). Thereafter, Petitioner filed updated medical records in March 2021, May 2021, and April 2022. Pet. Exs. 20-24.

In April 2022, the undersigned held a status conference where the parties agreed to be on a dual track toward resolution. Order dated Apr. 12, 2022 (ECF No. 73). This case was referred to Alternative Dispute Resolution ("ADR") on April 14, 2022. Order Referring Case to ADR dated Apr. 14, 2022 (ECF No. 74). And on April 22, 2022, the parties agreed to resolve entitlement through a ruling on the record. Pet. Status Rept., filed Apr. 22, 2022 (ECF No. 76).

Petitioner filed updated medical records in June 2022 while in ADR. Pet. Ex. 25. The parties remained in ADR until October 12, 2022. Order Concluding ADR Proceedings dated Oct. 12, 2022 (ECF No. 89).

Petitioner filed her motion for a ruling on the record on July 6, 2022. Pet. Motion for Ruling on the Record ("Pet. Mot."), filed July 6, 2022 (ECF No. 81). Respondent filed his responsive brief on November 15, 2022. Resp. Response to Pet. Mot. ("Resp. Response"), filed Nov. 15, 2022 (ECF No. 93). Petitioner filed her reply on December 19, 2022. Pet. Reply Brief for Ruling on Record ("Pet. Reply"), filed Dec. 19, 2022 (ECF No. 94).

This matter is now ripe for adjudication.

B. Factual History

1. Medical History

Prior to vaccination, Petitioner's medical history was significant for chronic back pain, depression, insomnia, obesity, restless leg syndrome, and sciatica. Resp. Rept. at 2 (citing Pet. Ex. 4 at 6, 87; Pet. Ex. 5 at 83, 332; Pet. Ex. 7 at 2-3, 25). Petitioner tested positive for hepatitis B surface antibody[3] and negative for hepatitis B core antibody[4] and hepatitis B surface antigen[5] in 2012.[6] Pet. Ex. 11 at 7.

On April 1, 2015, Petitioner received a lumbar injection for sciatica. Pet. Ex. 8 at 8-13. On April 7, 2015, she presented to Dr. Christopher B. Nicora to reestablish primary care. Pet. Ex. 20 at 12. Physical examination was normal, including deep tendon reflexes of 2+, intact sensory, and Babinski downgoing bilaterally. Id. On April 9, 2015, Petitioner reported breathing problems in a call to her treating physician. Pet. Ex. 7 at 37.

---

[3] A positive hepatitis B surface antibody result indicates protection against the hepatitis B virus that "can be the result of receiving the hepatitis B vaccine or successfully recovering from a past hepatitis B infection. . . . A positive . . . test result means you are 'immune' and protected against the hepatitis B virus and cannot be infected." Hepatitis B Blood Tests, Hepatitis B Found., https://www.hepb.org/prevention-and-diagnosis/diagnosis/hbv-blood-tests/ (last visited June 28, 2023).

[4] If a person tested positive for hepatitis B core antibody, that result would indicate a past or current hepatitis B infection. See supra note 3.

[5] If a person tests positive for hepatitis B surface antigen, they are infected with hepatitis B and the hepatitis B virus is present in their blood. See supra note 3.

[6] A positive test result for hepatitis B surface antibody and negative test results for the hepatitis B core antibody and hepatitis B surface antigen indicates a person is immune and protected. Understanding Your Test Results, Hepatitis B Found., https://www.hepb.org/prevention-and-diagnosis/diagnosis/understanding-your-test-results/ (last visited June 28, 2023).

On April 23, 2015, at 59 years of age, Petitioner received a hepatitis B vaccination as a requirement for her employment. Pet. Ex. 2 at 1; Pet. Ex. 3 at 1.

On May 4, 2015, Petitioner presented to the emergency department ("ED") at Putnam Hospital Center complaining of hand and leg weakness. Pet. Ex. 4 at 6. Petitioner "report[ed] that she woke up yesterday with onset of bilateral hand and leg weakness feeling like she 'couldn't use them properly.' [Petitioner] state[d] that she had a [c]orti[s]one shot administered about [three] weeks ago for sciatica." Id.; see also Pet. Ex. 8 at 8-13 (noting lumbar injection given on April 1, 2015). Petitioner "denie[d] fevers, chills, rashes, sore throat, congestion, shortness of breath, cough, chest pain, abdominal pain, nausea, vomiting, urinary symptoms, back pain, headache, dizziness[,] and any other medical complaints/concerns." Pet. Ex. 4 at 6. Dr. Freya J. Dittrich, D.O., conducted a physical examination that revealed bilateral upper and lower extremity strength of 5/5 "but fatigue[d] quickly" and "grip [slightly] weak but equal." Id. at 7. Laboratory results confirmed an acute urinary tract infection ("UTI"). Id. at 7-8. Diagnoses also included sinus tachycardia and generalized weakness. Id. at 8. Petitioner was prescribed antibiotics and discharged home. Id. at 8, 26.

Two days later, on May 6, 2015, Petitioner returned to Putnam Hospital Center ED and was seen by Dr. John Van Der Steeg "for evaluation of bilateral generalized weakness of the limbs." Pet. Ex. 4 at 87.

> [Petitioner] report[ed] that the weakness began in her hands and then spread to her legs. [Petitioner] was moving things around the house on Sunday (x3 days ago) when she noticed that she was unable to open, close, and hold objects as tightly as before. She was seen here on Monday (x2 days ago) with a similar complaint and was discharged home with a UTI and is currently taking [antibiotics] for it. [Petitioner] present[ed] back to the ED because she state[d] that the weakness ha[d] gotten worse. Today, [Petitioner] needed assistance putting on her clothes and getting to the chair. [Petitioner] came to the ED in a wheelchair.

Id. She "clarifie[d] that the weakness would not necessarily be classified as pain." Id. She also denied fever, rashes, urinary or bowel incontinence or changes, diarrhea, hematochezia, vision changes, and ear pain." Id. Physical examination noted "normal coordination observed, neurovascularly intact distally, 5/5 pedal push/pull bilaterally, good strength, . . . bilateral weak hand grasps, [and] [p]atellar reflexes 2 over 4." Id. at 89. GBS was listed under differential diagnosis. Id. A head computed tomography ("CT") was normal. Id. at 89-90.

Dr. Hillard Sharf conducted a neurology tele-medicine consultation that morning, on May 6. Pet. Ex. 4 at 77-78. History of present illness documented Petitioner's progressive weakness that began in the legs and moved to the hands, with "some numbness in feet and hands." Id. at 77. Petitioner was unable to walk without assistance and could not open a jar. Id. Dr. Sharf documented Petitioner had influenza ("flu") in February, some chronic lower back pain, and a recent epidural. Id. Dr. Sharf's impression stated, "Good story for [GBS]." Id. at 78. Dr. Sharf recommended Petitioner be admitted and obtain magnetic resonance imaging ("MRI") of lumbar and cervical spine, lumbar puncture, and electromyography ("EMG"). Id.

Petitioner was also seen by Dr. Anil Bhat that afternoon. Pet. Ex. 4 at 93. Dr. Bhat documented Petitioner's clinical course and noted her "hepatitis B vaccination last week." Id. Physical examination revealed "plantars downgoing[,] bilateral knee jerks symmetric, light touch[,] intact distal strength[,] and decreased feet hands intact biceps reflex." Id. at 94. Assessment was "probable acute idiopathic demyelinating polyneuropathy" and GBS. Id. For further treatment, Petitioner was transferred to Westchester Medical Center. Id. at 85, 90, 93; see also Pet. Ex. 5 at 22; Pet. Ex. 6 at 1-6. Diagnosis on transfer was GBS. Pet. Ex. 4 at 85.

On arrival at Westchester Medical Center on May 6, Petitioner was evaluated by Dr. Jonathan Berkowitz. Pet. Ex. 5 at 23-24. Triage note stated that Petitioner reported she "had a hep[atitis] [B] shot 1-1.5 [weeks] ago then [four] days ago started to exp[erience] bilat[eral] hand grip weakness and diff[iculty] walking [related to] weakness." Id. at 25. Dr. Berkowitz wrote Petitioner "fe[lt] the symptoms may be related to the [h]epatitis B vaccine she received [one] month ago." Id. On examination, Petitioner had intact sensation bilaterally, 3/5 strength in all extremities with distal worse than proximal, and 2+ reflexes bilaterally. Id. at 26. Clinical impression was GBS and Petitioner was admitted. Id. at 26-27.

Petitioner was seen by neurologist Dr. Noorie Pednekar on May 6, 2015. Pet. Ex. 5 at 47-50. Dr. Pednekar documented Petitioner's clinical course of bilateral weakness beginning Sunday (May 3, 2015). Id. at 47. Petitioner reported her "weakness continued to progress during the course of two days [and] now involve[ed] her [bilateral] [lower extremities] to the point that she had to go down the stairs sitting." Id. Petitioner "also developed isolated pain in her [bilateral] calves on [S]unday and is tender to touch now, however[,] [she] denie[d] pain anywhere else." Id. No associated complaints were reported. Id. History of viral illness in February and epidural injection in April were documented. Id. at 47-48. Physical examination revealed strength of 4/5 in finger flexion and 3/5 in finger extension and decreased reflexes of 1+ at ankles. Id. at 49. Assessment was acute onset progressive distal extremity weakness, with a note to rule out GBS. Id. EMG and lumbar puncture[7] were ordered. Id.

On May 7, 2015, Petitioner saw neurologist Dr. Stephen Marks. Pet. Ex. 5 at 96. He noted Petitioner had "[four] days of progressive weakness; [one] week after [h]ep[atitis] B vaccination." Id. Petitioner was "quadriparetic, distal slightly more than [proximal]" and normal deep tendon reflexes "except 1/4 left brach and absent [ankle jerks]." Id. His impression was polyneuropathy with a note to rule out certain conditions including GBS. Id. He ordered an EMG, and if it was consistent with GBS, then Petitioner was to begin intravenous immunoglobulin ("IVIG"). Id. Dr. Anila Thomas conducted an EMG/nerve conduction study ("NCS") later that day and found the results "consistent with a primarily demyelinating motor neuropathy," with some changes on EMG that "may be related to pre-existing pathology." Id. at 43. Petitioner was started on IVIG for five days. Id. at 92, 99. "[Petitioner's] weakness did improve but she was not back to her baseline." Id. at 317. Physical therapy evaluation found Petitioner to be a good candidate for acute rehabilitation. Id.

[7] Dr. Pednekar was unable to collect cerebrospinal fluid ("CSF") from the lumbar puncture done on May 7, 2015. Pet. Ex. 5 at 162.

Petitioner was discharged from Westchester Medical Center the morning of May 12, 2015 into the care of Acute Rehabilitation. Pet. Ex. 5 at 150, 333; Pet. Ex. 6 at 7-10. Discharge summary noted Petitioner was given a hepatitis B vaccination "recently." Pet. Ex. 5 at 332. Petitioner remained in acute rehabilitation until her discharge on May 19, 2015. Id. at 649. While in acute rehabilitation, she received physical and occupational therapy for up to three hours per day, five days a week. Id. at 650. On discharge, physical examination revealed 4/5 strength in right upper extremity and bilateral ankle dorsiflexion. Id. at 649-50. Petitioner was independent with activities of daily living and ambulation prior to discharge. Id. at 650.

On June 2, 2015, Petitioner had an initial occupational therapy evaluation at Putnam Hospital Center. Pet. Ex. 4 at 196. Petitioner reported bilateral hand weakness, decreased endurance, and decreased coordination. Id. She attended a total of four outpatient occupational therapy sessions between June 2 and June 18, 2015. Id. at 210-11.

Petitioner saw her primary care physician, Dr. Nadia Nord, on August 6, 2015. Pet. Ex. 7 at 39. Petitioner reiterated her clinical course and GBS diagnosis. Id. History of present illness documented "[Petitioner] had hep[atitis] b vaccine ~1-2 week[s] prior to all symptoms." Id. Petitioner reported "improvement in leg and arm weakness, but still [had] hand weakness and unsteady gait." Id. Neurology examination revealed bilateral lower extremity strength of 5-/5, bilateral upper extremity strength of 4+/5 except for hand/wrist strength of 3/5, and unsteady gait. Id. at 40.

Petitioner began physical therapy and occupational therapy in August 2015. Pet. Ex. 4 at 223, 238. She continued to have "difficulty walking, going up and down stairs[,] and dressing/[activities of daily living] due to hand weakness." Id. at 223. Her physical therapist documented that "[Petitioner] note[d] she had hepatitis injection this spring for a new job and two weeks later her symptoms started. [Petitioner] note[d] she also had a flu this winter." Id. Petitioner's occupational therapist documented Petitioner had "hepatitis B vaccination for work and developed [GBS] on May 3, 2015." Id. at 238. Petitioner attended additional physical therapy and occupational therapy sessions until November 2015. Id. at 223-68.

On October 19, 2015, Petitioner presented to Dr. Thomas at Westchester Medical Center for a neurology follow-up appointment. Pet. Ex. 5 at 664. Petitioner reportedly had a repeat EMG in July 2015[8] that was "consistent with a motor neuropathy with both demyelinating and axonal features. Compared to the prior study in May 2015, there [was] now evidence of axonal loss on both nerve conduction and EMG. In the arms[,] there [was] evidence of active denervation on EMG, without evidence of re-innervation . . . ." Id. at 665. Petitioner reported worsening sciatica pain since her GBS. Id. Physical examination revealed strength of 4/5 in biceps and triceps bilaterally, 4/5 in wrist flexion and extensor bilaterally, and 3/5 hand grip. Id. at 665-66. Deep tendon reflexes were 2+ throughout, equivocal Babinski, and positive straight leg test bilaterally. Id. at 666. Dr. Thomas found Petitioner was "recovering but slowly as expected in GBS." Id. Additionally, Dr. Thomas determined Petitioner's sciatica was "[l]ikely exacerbated due to immobility during hospital stay and weakness of stabilizing muscles." Id.

[8] The undersigned did not find a record of the EMG report filed.

Petitioner underwent an internal medicine evaluation with Dr. George Wootan for her state disability claim on October 26, 2015. Pet. Ex. 20 at 205. Petitioner reiterated her clinical course and reported having "a tremor, limited use of her hands, weakness, muscle wasting, and a lot of pain." Id. Petitioner also reported "she ha[d] to use a cane because she [was] unsteady" and "ha[d] fallen twice." Id. Additionally, her back pain had returned. Id. On examination, "[s]he walk[ed] flexed very steadily;" she was "very unsteady" without the cane; "[s]he could not walk on her heels or toes;" "[s]he only had [five] degrees of the squat;" and she had a wide stance. Id. at 206. Dr. Wootan noted Petitioner used her cane "for stability and to help take pressure off," and he found the cane was medically necessary. Id. Petitioner also needed assistance changing for examination and getting on and off the examination table, but she had minimal difficulty getting out of the chair. Id. Neurologic examination revealed absent deep tendon reflexes in all extremities, no sensory deficits, 3/5 strength in hands bilaterally, and 5/5 strength in lower extremities. Id. at 207. Dr. Wootan also noted Petitioner had muscle atrophy in between the thumb and forefinger bilaterally, Petitioner could not touch her thumb to her fifth digit bilaterally, and Petitioner's grip strength was 2/5 bilaterally. Id.

Petitioner returned for a follow-up examination on November 30, 2015 with Dr. Thomas. Pet. Ex. 5 at 671. Bilateral hand muscle atrophy was observed on examination. Id. at 672. Petitioner reported she would try taking gabapentin although she was concerned about weight gain. Id. at 673.

In December 2015, Petitioner moved and sought additional occupational therapy treatment at Montefiore. Pet. Ex. 7 at 57. History of present illness documented "[Petitioner] had hepatitis B vaccination [two] weeks before ([April 23, 2015]) she developed GBS." Id. By February 2016, Petitioner reported a 50% improvement in her symptoms, but she had continued difficulty with certain activities of daily living, including opening jars/bottles and using zippers. Id. at 109. She had normal strength throughout except in the hand intrinsic muscles (3/5). Id. at 110-11. Her lower back pain was worsening and she received an injection on February 29, 2016. Id. at 111; Pet. Ex. 8 at 15.

Moving forward to mid-2016, repeat EMG/NCS on May 24, 2016 revealed Petitioner's "[acute inflammatory demyelinating polyneuropathy ("AIDP")] ha[d] improved . . . and less active denervation [was seen] on EMG study." Pet. Ex. 5 at 678. On July 11, 2016, Petitioner saw neurologist Dr. Shyla Kodi at Westchester Medical Center. Pet. Ex. 5 at 682. Petitioner ambulated with a walker. Id. at 683. She reported "much improvement." Id. "[H]er ability to write ha[d] returned" and her overall motor strength improved, but she continued to complain of gait instability. Id. Petitioner was still taking gabapentin. Id. On examination, she had atrophy of intraosseous hand muscle in the left more than the right hand. Id. at 683-84. Petitioner was directed to continue with occupational and physical therapy as well as gabapentin. Id. at 684.

At a follow-up examination in March 2017, Petitioner was no longer using a walker to ambulate. Pet. Ex. 5 at 687. She was "able to walk [with] little assistance from a cane." Id. She reported new onset cramps in her lower extremities, and left upper extremity shoulder pain, but denied any new weakness. Id. Because physical examination revealed slightly decreased strength, Dr. Thomas ordered a repeat EMG/NCS. Id. at 688. Dr. Thomas also increased Petitioner's gabapentin. Id.

Petitioner saw physical medicine and rehabilitation specialist Dr. Anna Maria Lasak on September 12, 2016. Pet. Ex. 7 at 118. History of present illness documented Petitioner "had [flu] A in April 2015, followed by [h]ep[atitis] B vaccination, and subsequent GBS." Id. Petitioner completed occupational therapy and reported approximately 50% improvement in symptoms of weakness in hands. Id. She reported continued difficulties with walking and balance. Id. She also noted worsening lower back pain since her GBS diagnosis and she ambulated with a cane due to the chronic lower back pain. Id. Petitioner continued to take gabapentin. Id. at 119-21. Dr Lasak ordered more physical therapy. Id. at 121.

On June 29, 2017, Petitioner had a repeat EMG/NCS, which was "consistent with an improving polyneuropathy . . . now [with] predominantly motor involvement with axonal features." Pet. Ex. 5 at 690. Compared to the 2016 study, "further improvement in motor and sensory amplitudes, conduction velocity[,] and distal latency" were noted. Id.

Petitioner saw Dr. Marks for follow-up examination on March 28, 2019. Pet. Ex. 21 at 1. Physical examination revealed slightly decreased strength. Id. at 2. Petitioner also had difficulty walking on her toes. Id. He noted Petitioner "ha[d] a slightly wide-based gait with feet splayed outwards" and "[h]er turn involve[d] frequent steps and [was] not an adroit spin." Id. at 3. Assessments included "[GBS] following vaccination." Id. She returned to Dr. Marks on September 12, 2019, reporting that gabapentin was helpful in promoting sleep, but it did not help in decreasing muscle cramps in her calves at night. Id. at 5. Physical examination remained mostly unchanged, and the primary assessment remained "[GBS] following vaccination." Id. at 6-7.

On October 2, 2020, Petitioner had a follow-up examination with Dr. Thomas. Pet. Ex. 21 at 9. Petitioner reported continued cramping at night and when doing activities. Id. She did not have numbness or tingling, and she was no longer taking gabapentin. Id. Dr. Thomas summarized that "[t]he year [Petitioner] had GBS[,] she had the flu shot in October. Then she had the [f]lu on March 3rd and got Tamiflu. She had the hepatitis B shot in April. She developed GBS on May 6th." Id. Dr. Thomas stated,

> The GBS has been attributed to vaccination, although also could have been from the flu. . . . Here[,] her flu shot was too early to be related to GBS. So it would be linked to the flu or [h]epatitis B shot. If GBS were related to vaccination; we generally recommend holding off on that vaccination.

Id. Assessment was "[GBS] following vaccination." Id. at 10. On February 3, 2021, Petitioner returned to Dr. Thomas to discuss the Covid-19 vaccine as she had not received a vaccine in the past five years. Id. at 12.

In March and May 2022, Dr. Thomas provided letters stating Petitioner was "unable to work due to her neurologic symptoms from her history of [GBS]." Pet. Ex. 24 at 1; Pet. Ex. 25 at 1.

No additional relevant medical records were filed.

2. Affidavits

a. Petitioner

Prior to the vaccination at issue, Petitioner was hired by Arms Acres and advised that she needed to obtain a hepatitis B vaccination for her employment. Pet. Ex. 1 at ¶ 5. On April 23, 2015, she received the hepatitis B vaccination at issue. Id. at ¶ 7.

Around May 2 or 3, 2015, Petitioner's hand and leg weakness began, and by May 4, she presented to the ED at Putnam Hospital Center. Pet. Ex. 1 at ¶ 7. She was discharged with a diagnosis of a UTI, but her symptoms continued to worsen. Id. On May 6, 2015, Petitioner "could not walk and could not use [her] hands properly." Id. at ¶ 8. Petitioner was readmitted to Putnam Hospital Center that day. Id. Petitioner spoke to a neurologist, and "[a]fter he asked [Petitioner] about whether [she] received any vaccinations recently and [she] told him about the [h]epatitis B shot, he told [Petitioner] that he thought [she] had developed [GBS]." Id. at ¶ 9. Thereafter, Petitioner chose to transfer to a larger hospital, Westchester Medical Center, for testing. Id. at ¶¶ 9-10.

At Westchester Medical Center, Petitioner's GBS diagnosis was confirmed with an EMG and she began IVIG treatments. Pet. Ex. 1 at ¶ 11. Thereafter, Petitioner began rehabilitation at an in-patient facility. Id. at ¶ 12. After discharge, Petitioner continued with physical and occupational therapy; however, "[she] could not drive and was having difficulty finding rides to [her] appointments so [she] had to stop for a while." Id. at ¶ 13. Petitioner started therapy again multiple times throughout 2015, but had to stop due to insurance issues and a move. Id. at ¶¶ 14, 16.

Petitioner stated that "[a]lthough [she] had some back issues prior to the vaccination, [her] back pain has been significantly worse since this all started." Pet. Ex. 1 at ¶ 15. Her neurologist advised that "[her] back condition was likely exacerbated due to [her] lengthy immobility and due to the increased weakness of the muscles in and around [her] back after the GBS." Id.

As of April 19, 2018, the date on which she executed her affidavit, Petitioner "still ha[d] some significant symptoms and ha[d] not returned to the person that [she] was before the vaccination." Pet. Ex. 1 at ¶ 17. Petitioner still had issues walking and with her hands, affecting her writing, typing, sewing, cutting food, and using aerosol cans. Id. at ¶ 18. She occasionally experienced numbness and averred that the cold weather exacerbated her condition. Id. Since her GBS hospitalization, she has not worked and has been receiving Social Security Disability benefits. Id. at ¶ 19.

b. Daniel M. Drebycz

Mr. Drebycz is Petitioner's son. Pet. Ex. 9 at ¶ 2. In May 2015, Petitioner called Mr. Drebycz and indicated she could not walk. Id. at ¶ 5. He met her at Putnam Hospital Center. Id. at ¶ 6. At that time, Petitioner was unable to move her legs or squeeze Mr. Drebycz's hands. Id. "[O]ne of the first questions that [the neurologist] asked [Petitioner] was whether she received a

flu shot or other vaccination recently. When [Petitioner] told him about receiving the [h]epatitis B shot and told him when she had received it, the doctor then told [Petitioner and Mr. Drebycz] that he thought [Petitioner] had [GBS] . . . ." Id. at ¶ 7.

Once at Westchester Medical Center, Mr. Drebycz stated that "it seemed to be the consensus among all the doctors and staff that the [h]epatitis B shot was what caused [Petitioner] to develop GBS." Pet. Ex. 9 at ¶ 9.

As of the date he executed his affidavit, April 19, 2018, Mr. Drebycz averred that Petitioner "does not walk the same and has continued difficulty with her fine motor skills and movements. She still does not drive and has moved back into the house with [his] grandfather as she cannot live independently." Pet. Ex. 9 at ¶ 10.

c. Anna Marie Antenucci

Ms. Antenucci met Petitioner over 20 years ago at work and they have remained friends in regular contact since. Pet. Ex. 10 at ¶¶ 2-3. She recalled Petitioner calling in early May 2015 stating that she was unable to walk or use her hands properly. Id. at ¶ 5. She visited Petitioner in the hospital. Id. at ¶ 6. And once she was discharged, Ms. Antenucci visited Petitioner "every day to help take care of her." Id. at ¶ 7. Ms. Antenucci averred "[she] saw first-hand how [Petitioner] would struggle walking and the difficulty she was having with her hands. [She] would help her with a number of everyday activities . . . [and] [she] also used to drive [Petitioner] to her rehabilitation appointments when [she] could." Id. at ¶ 8.

She stated that prior to the hepatitis B vaccination at issue, Petitioner did not have these issues. Pet. Ex. 10 at ¶ 9. Ms. Antenucci added that "[Petitioner] has always told [her] that the [h]epatitis B shot was what caused her to develop GBS and [Ms. Antenucci] also firmly believe[s] that as [she] [does not] know of anything else that happened to [Petitioner] around that time which could explain her sudden onset of symptoms." Id.

Ms. Antenucci averred that as of April 20, 2018, the date on which her affidavit was executed, "[Petitioner] does not walk the same and still has difficulty with her hands." Pet. Ex. 10 at ¶ 10. They no longer see each other daily; however, when they do see each other, Ms. Antenucci "notice[s] [Petitioner's] hands still sometimes shake and still sometimes look gnarled and [Petitioner] has difficulty doing certain things such as opening jars or cans." Id.

C. Expert Reports

1. Petitioner's Expert, Dr. Marcel Kinsbourne, M.D.[9]

a. Background and Qualifications

Dr. Kinsbourne is a neurologist, pediatrician, and pediatric neurologist. Pet. Ex. 18 at 1. In 1955, he obtained his B.M., B.Ch., from Oxford University Medical School, and he completed

[9] Dr. Kinsbourne provided two expert reports. Pet. Exs. 18-19.

postdoctoral training through 1964 in the United Kingdom. Id.; Pet. Ex. 27 at 1. Thereafter, he obtained board certification and licensing in the United States and Canada and worked as a Professor at various teaching institutions since 1967. Pet. Ex. 18 at 1; Pet. Ex. 27 at 1-2. Dr. Kinsbourne has served and is currently serving on a number of editorial boards. Pet. Ex. 27 at 3. He has authored or co-authored more than 400 publications. Id. at 5-33.

b. Opinion

i. Althen Prong One[10]

Dr. Kinsbourne opined that the hepatitis B vaccination can cause GBS through molecular mimicry. Pet. Ex. 18 at 3. First, he noted infections, both viral and bacterial, as well as vaccinations have been found to trigger GBS. Id.; see, e.g., Pet. Ex. 18.2 at 2 ("[GBS] is related in most cases to respiratory or gastrointestinal infectious and vaccines . . . ."); [11] Pet. Ex. 18.5 at 1 (acknowledging GBS has been reported in association with viral hepatitis, albeit rarely); [12] Pet. Ex. 26 at 1 (noting GBS has been reported following viral hepatitis and hepatitis B vaccination); [13] Pet. Ex. 19.1 at 1 (stating GBS has been found to occur after viral or bacterial infections as well as various vaccines). [14]

Next, he explained the process by which a vaccine can induce GBS via molecular mimicry. Pet. Ex. 18 at 3. Following vaccination, the innate immune system detects epitopes of invading organisms and launches an immune attack on such epitopes; however, this "attack" can sometimes turn against the structurally similar epitopes on an individual's own cells. Id. With GBS, "those target self-epitopes are gangliosides on myelin sheaths and axons." Id.

[10] In Petitioner's Motion, she states that "there have been approximately 275 [Vaccine Adverse Event Reporting System ("VAERS")] reports filed alleging the development of GBS after receiving the [h]epatitis B vaccine alone." Pet. Mot. at 17 (citing Pet. Ex. 15). The exhibit is a one page document with no reference or citation. Thus, it is unclear where Petitioner obtained this data. Additionally, Petitioner's expert, Dr. Kinsbourne, does not reference this document in his reports. The undersigned did not rely on this exhibit as she was unable to verify its origins or substantive content.

[11] M. Khamaisi et al., Guillain-Barré Syndrome Following Hepatitis B Vaccination, 22 Clinical & Experimental Rheumatology 767 (2004).

[12] Kidist K. Yimam et al., A Rare Case of Acute Hepatitis B Virus Infection Causing Guillain-Barré Syndrome, 9 Gastroenterology & Hepatology 121 (2013).

[13] A. Kakar & P.K. Sethi et al., Guillain Barre Syndrome Associated with Hepatitis B Vaccination, 64 Indian J. Pediatrics 710 (1997).

[14] Ebru Arhan et al., Guillain-Barre Syndrome Associated with Hepatitis B Vaccine and a Review of the Literature, 20 Gazi Med. J. 83 (2009).

Dr. Kinsbourne opined that "epidemiological evidence, [] individual case reports, [and] a background of hepatitis B infection being itself a cause of GBS . . . suffices as circumstantial evidence to establish [hepatitis B vaccine] causation of GBS as a medically reasonable cause of GBS." Pet. Ex. 18 at 3. For support, he cited several articles and case reports.

In 1988, Shaw et al.[15] examined 41 reports of adverse events following vaccination with a plasma-derived[16] hepatitis B vaccine over a period of three years. Pet. Ex. 18.3 at 1. Of those 41 reports, nine were GBS, and all nine occurred within seven weeks of the most proximate hepatitis B vaccination. Id. at 5-6. Five of the nine GBS patients experienced symptoms consistent with a viral illness within the four weeks prior to onset, and two of the nine received other vaccinations prior to onset. Id. at 6, 9 tbl.2. The authors found "[GBS] was reported significantly more often than expected," and using intervals of six and eight weeks, they found the incidence rate was "significantly higher" than the Centers for Disease Control and Prevention ("CDC") background rate. Id. at 8.

The authors in Shaw et al. addressed whether "it [is] biologically plausible that [GBS] could be caused by the hepatitis B vaccine." Pet. Ex. 18.3 at 13. They noted several reports of GBS post-hepatitis B infection and that GBS had been "anecdotally reported" following many vaccines. Id. The authors also questioned whether GBS was underreported, and whether this affected their findings because adverse event reports were collected by a passive reporting system. Id. Dr. Kinsbourne agreed that due to underreporting, these findings were "probably [] an underestimate of the effect of [hepatitis B vaccination] on the incidence of GBS." Pet. Ex. 18 at 3; see Pet. Ex. 18.3 at 1, 13. Shaw et al. concluded "no conclusive epidemiologic association could be made." Pet. Ex. 18.3 at 1.

After Shaw et al. was published, the CDC issued a Morbidity and Mortality Weekly Report ("MMWR"),[17] providing recommendations of the Advisory Committee on Immunization Practices ("ACIP"). Pet. Ex. 12. Relying on the findings in Shaw et al., the CDC stated that "[i]n the United States, surveillance of adverse reactions has shown a possible association between [GBS] and receipt of the first dose of plasma-derived hepatitis B vaccine." Id. at 11 (citing Pet. Ex. 18.3). In his supplemental expert report, Dr. Kinsbourne noted the recombinant hepatitis B vaccine "has been shown to be comparable to the plasma-derived vaccine in terms of animal potency . . . and protective efficacy." Pet. Ex. 19 at 1.[18] Thus, he believed there was no

[15] Frederic E. Shaw et al., Postmarketing Surveillance for Neurologic Adverse Events Reported After Hepatitis B Vaccination, 127 Am. J. Epidemiology 337 (1988).

[16] Specifically, the plasma-derived vaccine was manufactured from the plasma of hepatitis B virus carriers. Pet. Ex. 18.3 at 2.

[17] Ctrs. for Disease Control & Prevention, Hepatitis B Virus: A Comprehensive Strategy for Eliminating Transmission in the United States Through Universal Childhood Vaccination: Recommendations of the Immunization Practices Advisory Committee (ACIP), 40 Morbidity & Mortality Wkly. Rep., Nov. 22, 1991.

[18] The source of this information was not filed.

evidence "the recombinant vaccine claimed to be safer than the plasma-derived product . . . [n]or [was] there any support for the claim that a recombinant vaccine is less likely than a plasma-derived vaccine to cause autoimmune reactions such as GBS." Id.

In 2004, Geier and Geier[19] examined adverse events reported in literature from 1966 to 2003 and to the Vaccine Adverse Event Reporting System ("VAERS") database[20] following vaccination with hepatitis B.[21] Pet. Ex. 19.2 at 2. In the literature, they found eight reports of GBS following hepatitis B vaccination. Id. at 3 tbl.I. The median onset was six days. Id. In the VAERS database, they found 93 reports of GBS post-hepatitis B vaccination, with a median onset of 18 days. Id. at 3 tbl.II.

A few years later in 2007, Souayah et al.[22] examined VAERS reports of GBS following vaccination in 2004. Pet. Ex. 18.4 at 1. They found 54 cases of GBS reported after vaccination, and seven of the 54 were following only hepatitis vaccination. Id. at 1-2, 2 tbl.1. Six of the seven patients developed GBS within six weeks of vaccination, and the onset period was unknown in the seventh patient. Id. at 2, 2 tbl.1. Eleven of the 54 cases were following a combination of two or more vaccines, and five of the eleven received a hepatitis vaccination. Id. at 2 tbl.1. Although the authors did not specify which hepatitis vaccination was administered in these cases, they found the "[h]epatitis vaccine was the second most frequently associated vaccine with GBS." Id. at 2.

Souayah et al. acknowledged "[their] study ha[d] some limitations because it [was] based on data from VAERS, a passive surveillance system, where underreporting, differential reporting, ascertainment bias[,] and variability in report quality and completeness may occur;"

---

[19] M.R. Geier & D.A. Geier, A Case-Series of Adverse Events, Positive Re-Challenge of Symptoms, and Events in Identical Twins Following Hepatitis B Vaccination: Analysis of the Vaccine Adverse Event Reporting System (VAERS) Database and Literature Review, 22 Clinical & Experimental Rheumatology 749 (2004). This exhibit is missing one page containing the authors' findings and conclusions. Additionally, the undersigned notes that the Geier authors have been almost wholly discredited as experts in the Vaccine Program. See, e.g., America v. Sec'y of Health & Hum. Servs., No. 17-542V, 2022 WL 278151, at *8 n.16 (Fed. Cl. Spec. Mstr. Jan. 4, 2022) (listing cases that have discredited Geier and Geier); Chambers v. Sec'y of Health & Hum. Servs., No. 19-140V, 2022 WL 33693332, at *10 n.15 (Fed. Cl. Spec. Mstr. July 22, 2022) (noting "that the Geiers have repeatedly, and over a lengthy period of time, been deemed to be questionably-competent and scientifically-unreliable experts in the Vaccine Program—casting significant doubt on *any* studies they have authored"). As such, the undersigned does not rely on this article.

[20] For more information on VAERS, see Pet. Ex. 19.2 at 2.

[21] The article does not confirm whether the GBS patients received the plasma-derived or recombinant vaccine. See Pet. Ex. 19.2.

[22] Nizar Souayah et al., Guillain-Barre Syndrome After Vaccination in United States: A Report from the CDC/FDA Vaccine Adverse Event Reporting System, 25 Vaccine 5253 (2007).

however, they stressed their study objective was to identify the occurrence of GBS. Pet. Ex. 18.4 at 3. They concluded "[their] results suggest that vaccines other than [flu] vaccine can be associated with GBS." Id. Again Dr. Kinsbourne agreed these results may be affected by underreporting due to the passive reporting nature of VAERS. Pet. Ex. 18 at 3.

Dr. Kinsbourne also cited several case reports to support his theory. Kakar and Sethi (1997) discussed the case of a three-year-old child who developed GBS following hepatitis B vaccination. Pet. Ex. 26 at 1. Their patient received a recombinant hepatitis B vaccine, and the following day, she presented with "sudden onset of weakness in both lower limbs." Id. The authors determined their case "clinically resembled GBS although conduction studies showed motor and sensory involvement." Id. at 2. Because of the brief onset, Kakar and Sethi hypothesized that (1) the patient was already primed by the antigen and the vaccine acted as a trigger or (2) the "GBS was unrelated to the vaccine and was caused by a virus." Id. Kakar and Sethi briefly discussed other studies and reports examining the association between GBS and hepatitis B vaccination,[23] and they acknowledged that the Institute of Medicine ("IOM") "found the evidence inadequate to accept or reject a causal relation of GBS following hepatitis B vaccine." Id. Additionally, Kakar and Sethi noted molecular mimicry was a mechanism thought to cause demyelination in GBS because the DNA of hepatitis B virus was homologous with myelin basic protein. Id.

Next, Vital et al.[24] (2002) reported two patients who developed an inflammatory polyneuropathy after hepatitis B vaccination.[25] Pet. Ex. 19.6 at 1. One patient presented "with acute sensory disturbances in the lower limbs" 15 days following vaccination. Id. The second patient presented with "severe motor and sensory neuropathy involving all [four] limbs" 21 days after vaccination. Id. at 2. The authors briefly discussed other case reports of inflammatory polyneuropathies or GBS following hepatitis B vaccination.[26] Id. at 4. Both patients exhibited axonal lesions the authors thought were "probably due to an autoimmune mechanism directed at some axonal or neuronal components." Id. Vital et al. opined the "autoimmune reaction was

---

[23] In one study, the authors examined adverse effects of hepatitis B vaccination and found "no increase in the incidence of GBS in the vaccinees," and thus concluded "the vaccine [was] safe and adverse reactions, if at all, [were] coincidental." Pet. Ex. 26 at 2. Another was a case report of a patient who presented with paresthesias in all four extremities ten days after administration of a second hepatitis B vaccination and following a respiratory tract infection. Id. This patient did not meet the clinical criteria of GBS, and so the patient was assessed with "an inflammatory polyneuropathy." Id. And in another case report, a patient developed a polyneuropathy two weeks after receiving a hepatitis B vaccine. Id. None of these articles were filed in this case. Thus, it is difficult to verify the information or discern whether it is reliable.

[24] Vital et al., Postvaccinal Inflammatory Neuropathy: Peripheral Nerve Biopsy in 3 Cases, 7 J. Peripheral Nervous Sys. 163 (2002).

[25] This article does not confirm the type of hepatitis B vaccination administered. See Pet. Ex. 19.6.

[26] None of these reports were filed in this case.

probably triggered by [hepatitis B vaccination] and was followed mainly by a severe axonal degeneration, corresponding to an acute sensory ataxic neuropathy [("ASAN")] in case [one], and to an acute motor and sensory axonal neuropathy (AMSAN) in case [two]." Id. (internal citations omitted).

Khamaisi et al. (2004) discussed the case of a 52-year-old woman who developed GBS after her second dose of a recombinant hepatitis B vaccine. Pet. Ex. 18.2 at 1. Ten weeks after vaccination, she presented with abdominal pain as well as a two-week history of progressive muscle weakness and limb tenderness particularly in her legs. Id. She denied any infections in the previous ten weeks. Id. She was diagnosed with GBS and common infectious causes were ruled out. Id. Given the eight weeks between vaccination and onset, the authors found "[t]he temporal relationship . . . was suggestive of a vaccine-induced cause." Id.

In their case report, Khamaisi et al. noted vaccines, including the hepatitis B vaccine, have been reported to be related to GBS. Pet. Ex. 18.2 at 2. They reviewed 19 other cases[27] of GBS post-hepatitis B vaccination. Id. at 2, 2 tbl.1. Seventeen of the 19 had an onset of symptoms between one day and eight weeks. Id. at 2 tbl.1. In addition to their case, Khamaisi et al. noted eight other case reports of GBS post-hepatitis B vaccination, three of which were following a recombinant hepatitis B vaccination. Id. at 3.

Additionally, Khamaisi et al. described three mechanisms that have been suggested to trigger GBS, including molecular mimicry. Pet. Ex. 18.2 at 3. They explained, "[h]epatitis B surface protein may provoke an autoimmune attack on a similar protein present in the nerve cells. In molecular mimicry involving T lymphocytes[,] these cells recognize their antigen as peptide-bound to [major histocompatibility complex ("MHC")] molecule."[28] Id. They also noted, like Kakar and Sethi, that "[t]he DNA sequence of [hepatitis B virus] was found to be homologous to myelin basic protein." Id. Even though adverse reactions, such as GBS, are reported following vaccinations, the authors found "[t]hese reactions are sporadic and there is no clear evidence that the rate of GBS . . . is more common among the vaccinated population." Id.

In Arhan et al. (2009), the authors provided a case report of a 14-year-old male who developed GBS following hepatitis B vaccination as well as a review of similar literature. Pet. Ex. 19.1. Their patient received his first recombinant hepatitis B vaccination, and ten days later, developed muscle weakness and was unable to walk. Id. at 1-2. He had no history of upper respiratory tract infection, gastroenteritis, or fever. Id. at 1. Testing for certain infections, including cytomegalovirus, herpes, and Epstein-Barr virus, were negative, as well as serologic testing for *Campylobacter jejuni*. Id. at 1-2. Testing confirmed GBS diagnosis. Id. at 2.

---

[27] These 19 cases include the patients in Shaw et al. and Kakar and Sethi. See Pet. Ex. 18.2 at 2-3, 2 tbl.1 (citing Pet. Exs. 18.3, 26).

[28] Major histocompatibility complex is "the genes determining the major histocompatibility antigens, in all species a group of closely linked multiallelic genes located in a small region on one chromosome." Major Histocompatibility Complex, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=66341 (last visited June 28, 2023).

The Arhan et al. authors noted GBS has been reported following respiratory or gastrointestinal infections and vaccination, including the plasma-derived and recombinant hepatitis B vaccine. Pet. Ex. 19.1 at 2. A review of similar literature revealed over 20 cases[29] of GBS post-hepatitis B vaccination. Id. "Although the pathogenesis of hepatitis B vaccine associated GBS has not been completely elucidated, three mechanisms have been proposed," including "[m]olecular mimicry between viral antigens and neural host tissues." Id. Similar to Kakar and Sethi and Khamaisi et al., Arhan et al. noted "[t]he DNA sequence of [hepatitis B virus] was found to be homologous to myelin basic protein." Id.

Lastly, Dr. Kinsbourne cited Yimam et al., which discussed a case of GBS following hepatitis B viral infection. Pet. Ex. 18.5 at 1. Yimam et al. reported a case of GBS in a 42-year-old woman following a "[two]-week history of nausea, vomiting, and abdominal pain and a [one]-week history of jaundice." Id. GBS diagnosis was confirmed and it "was thought to be secondary to acute [hepatitis B viral] infection." Id. at 2. Yimam et al. also noted "[s]everal mechanisms have been proposed to explain how [hepatitis B virus] causes GBS," including "molecular mimicry between [hepatitis B virus] DNA and myelin basic protein, whereby initial host immunity to [hepatitis B virus] leads to the subsequent antibody-mediated attack of the myelin sheath." Id. at 3. The authors acknowledged that "[a]lthough the exact pathophysiology of GBS related to acute [hepatitis B virus] infection remains unclear, the association of these [two] conditions is well documented, as seen in this case report as well as earlier case reports." Id.

In response to Respondent's expert Dr. Moulton's opinion that there is no statistical or epidemiologic evidence supporting a finding that more likely than not the hepatitis B vaccine can cause GBS, Dr. Kinsbourne opined that "[e]pidemiology cannot be relied on to uncover rare events, vaccinal or otherwise. Instead, rare but potentially significant discoveries in medicine are contributed by observing individual patients and publishing the events in case reports." Pet. Ex. 19 at 3. For support, he cited Harper et al.[30] and Vandenbroucke,[31] who both noted the importance of case reports and case series on the progress of medicine, as they are the first to identify a problem. Id. (citing Pet. Exs. 19.3, 19.5). Dr. Kinsbourne added that "[c]ontrolled studies of sufficient scale of any rare events are hard to come by[] because they require vast numbers of control subjects, calling for an investment in effort and resources disproportionate to their impact on public health" and "[s]mall-scale studies risk false negative outcomes." Id. at 4. He concluded that a "lack of epidemiological support for causation of a rare event does not disqualify a petitioner from a positive ruling in the vaccine injury compensation program, particularly if there is supporting circumstantial evidence," as there is here. Id. at 2.

---

[29] These 20 cases include the patients in Shaw et al., Kakar and Sethi, and Khamaisi et al. Pet. Ex. 14 at 2 (citing Pet. Exs. 18.2-18.3, 26).

[30] Diane M. Harper et al., Review of Gardasil, 1 J. Vaccines & Vaccination 1 (2010).

[31] Jan. P. Vandenbroucke, In Defense of Case Reports and Case Series, 134 Annals Internal Med. 330 (2001).

ii. Althen Prongs Two and Three

Dr. Kinsbourne opined "to a reasonable degree of medical probability" and "more likely than not," Petitioner's GBS was caused by her hepatitis B vaccination. Pet. Ex. 18 at 4; Pet. Ex. 19 at 4.

Dr. Kinsbourne summarized Petitioner's clinical course. Pet. Ex. 18 at 1-2. Petitioner received a hepatitis B vaccine on April 23, 2015. Id. at 1. On May 4, she presented to the ED complaining of a two-day history of hand and leg weakness. Id. at 2. Two days later, she returned to the ED for increased weakness. Id. Petitioner had decreased strength and deep tendon reflexes at the ankles. Id. An EMG was performed on May 7 and confirmed diagnosis of GBS. Id.

He found no evidence of any other viable cause of Petitioner's GBS other than hepatitis B vaccination. Pet. Ex. 18 at 4. Dr. Kinsbourne disagreed with Respondent's expert Dr. Leist's opinions as to alternative causation. Pet. Ex. 19 at 2. First, Dr. Leist cited to Petitioner's complaint of a breathing problem on April 9, 2015. Id. (citing Resp. Ex. C at 6). Dr. Kinsbourne noted this entry was the only documentation of such complaint in Petitioner's medical records. Id. (citing Resp. Ex. C at 6).

Next, Dr. Leist opined it is "possible" that Petitioner had upper respiratory symptoms in the weeks before her GBS onset. Pet. Ex. 19 at 2 (quoting Resp. Ex. C at 6). However, Dr. Kinsbourne found no medical record evidence to support such an opinion, and instead suggested that "it [was] equally possible that [Petitioner] had allergic rhinorrhea." Id.

Lastly, Dr. Kinsbourne did not agree with Dr. Leist's argument that Petitioner had the flu in April 2015, which could have been an alternative cause of her GBS. Pet. Ex. 19 at 2. He explained that Petitioner saw two different health care providers and made various phone calls to her treating physicians in April 2015, and none of those records indicated that she had complaints of the flu or a flu-like illness, or that she was diagnosed with the flu. Id. Instead, Dr. Kinsbourne asserted that Petitioner's May 2015 records documented that Petitioner reported having the flu in February 2015 and being prescribed Tamiflu. Id. Thus, he concluded "the contemporaneous medical records do not support a diagnosis of . . . the flu in April 2015[] or that an upper respiratory infection caused her GBS." Id.

With regard to onset, Dr. Kinsbourne found Petitioner developed symptoms consistent with GBS nine days after receiving a hepatitis B vaccination, which he opined was "within the risk interval for GBS[,] and thus medically reasonable." Pet. Ex. 18 at 4. The articles and case reports cited by Petitioner support an onset period of one day to eight weeks post-hepatitis B vaccination.

Shaw et al. examined nine cases of GBS following hepatitis B vaccination and all nine occurred within seven weeks of vaccination. Pet. Ex. 18.3 at 5-6. Six of the seven reports in Souayah et al. developed GBS within six weeks of a hepatitis vaccination. Pet. Ex. 18.4 at 2, 2 tbl.1.

The patient in Kakar and Sethi developed GBS one day after hepatitis B vaccination. Pet. Ex. 26 at 1. The patients in Vital et al. had onset periods of 15 and 21 days post-hepatitis B vaccination. Pet. Ex. 19.6 at 1-2. Khamaisi et al. reported a case of GBS eight weeks post-hepatitis B vaccination. Pet. Ex. 18.2 at 1. In Arhan et al., the patient developed GBS 10 days after vaccination with hepatitis B. Pet. Ex. 19.1 at 1-2.

Lastly, the patient in Yimam et al. developed GBS within two weeks of hepatitis B infection. Pet. Ex. 18.5 at 1-2.

2. Respondent's Expert, Lawrence Moulton, Ph.D[32]

a. Background and Qualifications

Dr. Moulton is a biostatistician with experience as the principal statistician on numerous vaccine studies, including Haemophilus influenzae type b and pneumococcal vaccine trials. Resp. Ex. A at 1. He obtained a B.A. in statistics and mathematics from State University of New York at Buffalo, an M.S. in biometry from the University of Texas School of Public Health, and a Ph.D. in biostatistics from Johns Hopkins University. Resp. Ex. B at 1. He is currently a Professor in the Department of International Health and Department of Biostatistics at Johns Hopkins University as well as the Co-Director of the Institute for Vaccine Safety at Johns Hopkins University. Id. Dr. Moulton serves on Data Safety Monitoring Boards for over a dozen vaccine products, has advised the World Health Organization ("WHO") on various vaccine issues, and has over 200 publications, mostly concerning vaccines or infectious diseases. Resp. Ex. A at 1.

b. Opinion[33]

Dr. Moulton did not offer medical opinions, and instead focused his report on the medical literature cited by Dr. Kinsbourne and statistical and epidemiologic aspects of the literature. See Resp. Ex. A at 1-4. He opined the evidence does not support a finding that "more likely than not" the hepatitis B vaccine can cause GBS, or that the hepatitis B vaccine caused Petitioner's GBS. Id. at 4.

First, with regard to Shaw et al., Dr. Moulton argued the study had "caveats." Resp. Ex. A at 2. First, the number of people vaccinated with hepatitis B during the study period was not known. Id. Second, he noted that seven of the nine reported cases of GBS were from health care professionals "who may have [had a] much higher daily exposure to other infectious agents than

[32] Dr. Moulton provided one expert report. Resp. Ex. A.

[33] Dr. Mouton's report discusses "estimations" and "assumptions" concerning vaccination rates. Resp. Ex. A at 2-4. For example, he used a "complicated estimation procedure, with several assumptions, [and] came up with the number 838,215 [for number of people vaccinated during a certain period of time], based on a total of 2,417,000 doses delivered." Id. at 2. The undersigned does not address these aspects of his report due to the inability to verify these numbers or to determine the reliability of them.

the general population."[34] Id. And "[i]n those first few years, evidently 80-90% of hepatitis B vaccine recipients were health care professionals," and "thus, [] was a highly select group." Id. Third, the background incident rates used were not among health care workers. Id. He hypothesized that "if their GBS rates were [three] times higher than the general population . . . , that would eliminate the estimates of excess risk reported in Shaw et al." Id. Lastly, Dr. Moulton noted Shaw et al. concluded that there was "no conclusive epidemiologic association [that] could be made between any neurologic adverse event and the vaccine." Id.

Dr. Moulton also discussed the article by Souayah et al. Resp. Ex. A at 2. He first noted that no distinction was made between the hepatitis A vaccine and the hepatitis B vaccine. Id. Again he argued the reporting rates following these vaccines were not known. Id. at 2-3. He agreed with Dr. Kinsbourne that "underreporting is a well-known feature of passive reporting systems such as VAERS," but argued "it is not clear whether there is more underreporting when given [flu] vs. hepatitis vaccines." Id. at 3. He hypothesized "that since [flu] vaccines are so ubiquitous[,] [] problems following their receipt are less often reported to VAERS." Id. Dr. Moulton concluded that "[g]iven the vagaries of VAERS reporting, this publication should not be viewed as giving convincing support to the theory of GBS being caused by [the] hepatitis B vaccine." Id.

Next, Dr. Moulton addressed the case reports of Kakar and Sethi and Arhan et al. Resp. Ex. A at 3. He opined that a one-day onset in Kakar and Sethi supports a finding that such occurrence was not "far-fetched" and that such "coincidences" happen often. Id. Regarding the Arhan et al. patient, Dr. Moulton maintained that the case "implied co-existence, not causality." Id.

Dr. Moulton cited to the 2012 IOM report that concluded "[t]he evidence [was] inadequate to accept or reject a causal relationship between hepatitis B vaccine and GBS." Resp. Ex. A, Tab 2 at 2.[35] He noted that the IOM examined the studies Dr. Kinsbourne cited, including Kakar and Sethi, Khamaisi et al., and Souayah et al., when reaching their findings. Resp. Ex. A at 3 (citing Resp. Ex. A, Tab 2 at 1-2; Pet. Exs. 18.2, 18.4, 26). However, as Dr. Moulton noted, the IOM did not consider Souayah et al. "in the weight of epidemiologic evidence because they provided data from passive surveillance systems and lacked unvaccinated comparison populations." Id. (quoting Resp. Ex. A, Tab 2 at 1). When considering the mechanistic evidence, he explained the IOM determined Kakar and Sethi and Khamaisi et al. "did not provide evidence beyond temporality, some too long or too short based on the possible mechanisms involved." Id. (quoting Resp. Ex. A, Tab 2 at 2). Dr. Moulton failed to address or counter the IOM's statement that "molecular mimicry may contribute to the symptoms of GBS." Resp. Ex. A, Tab 2 at 2.

---

[34] For example, Dr. Mouton noted "antecedent flu-like illness has been reported to impart a 7-fold increase in GBS incidence in an Italian case-control study." Resp. Ex. A at 2 (citing Resp. Ex. A, Tab 1 (Guillain-Barré Syndrome Study Group, Guillain-Barré Syndrome: An Italian Multicentre Case-Control Study, 21 Neurological Scis. 229 (2000))).

[35] Inst. of Med., Hepatitis B Vaccine, in Adverse Effects of Vaccines: Evidence and Causality 435 (Kathleen Stratton et al. eds., 2012).

Dr. Moulton concluded Dr. Kinsbourne's literature "contained little if any evidence bearing on causality (in the statistical or epidemiologic frameworks)" between hepatitis B vaccination and development of GBS. Resp. Ex. A at 4. He suggested that Petitioner's evidence supports a finding that GBS cases can be expected to occur after vaccination "by chance alone." Id. at 3-4. And he maintained Petitioner's evidence "certainly does not provide evidence (in the statistical or epidemiologic frameworks) that would support a legal determination that it is 'more likely than not' that either hepatitis B vaccine can cause GBS, or that in this one particular situation, that hepatitis B vaccine caused the claimed GBS condition of the [P]etitioner." Id.

3. Respondent's Expert, Dr. Thomas P. Leist, M.D., Ph.D.[36]

a. Background and Qualifications

Dr. Leist is a board-certified neurologist, specializing in the field of neuroimmunology and is "regularly involved in the care of patients with neuroimmunological conditions including multiple sclerosis, transverse myelitis, neuromyelitis optica, [neuromyelitis optica] syndrome, and immune disorders of the peripheral nervous system." Resp. Ex. C at 1. He received a Ph.D. in biochemistry from the University of Zurich in Switzerland and an M.D. from the University of Miami in Florida, and after, he completed an internal medicine internship at the University of Miami and a neurology residency at Cornell Medical Center/Sloan Kettering Memorial Cancer Center in New York. Resp. Ex. D at 1. Dr. Leist currently works as a neurology professor at Thomas Jefferson University and holds multiple hospital and administrative appointments. Id. Dr. Leist has authored or co-authored various publications on the subject of immunology, neuroimmunology, and imaging. Id. at 6-11; Resp. Ex. C at 1.

b. Opinion

Dr. Leist opined the hepatitis B vaccine has not been found to be causally associated with GBS and the vaccine did not cause Petitioner to develop GBS. Resp. Ex. C at 5-7.

i. Althen Prong One

Dr. Leist did not address or refute Dr. Kinsbourne's theory of molecular mimicry. See Resp. Ex. C at 1-7. He also did not discuss or rebut Dr. Kinsbourne's cited medical literature that discussed homology between the hepatitis B virus and myelin basic protein. See, e.g., Pet. Exs. 18.2, 19.1, 26.

Dr. Leist opined the hepatitis B vaccine has not been shown to cause GBS. Resp. Ex. C at 5, 7. For support, he noted the IOM (now the National Academy of Medicine), the CDC, and the WHO have found no association between the hepatitis B vaccine and GBS. Id.

First, the IOM report from 2012 found "[t]he evidence [was] inadequate to accept or reject a causal relationship between hepatitis B vaccine and GBS." Resp. Ex. A, Tab 2 at 2.

[36] Dr. Leist provided one expert report. Resp. Ex. C.

Additionally, Dr. Leist reported that the epidemiologic evidence was found to be "insufficient or absent" and the mechanistic evidence was "lacking." Id. And when coming to these conclusions, Dr. Leist opined that the IOM reviewed the same literature cited by Dr. Kinsbourne, including Souayah et al., Kakar and Sethi, and Khamaisi et al. Resp. Ex. C at 6-7 (citing Resp. Ex. A, Tab 2 at 1-2; Pet. Exs. 18.2, 18.4, 26). Dr. Leist explained that the Souayah et al. authors relied on data from VAERS, which "is a passive surveillance system set up to detect possible safety signals" that "cannot serve to determine incidence rates or assign causality." Id. at 6. Dr. Leist failed to address or counter the IOM's statement that "molecular mimicry may contribute to the symptoms of GBS." Resp. Ex. A, Tab 2 at 2.

Next, Dr. Leist cited to the CDC Pink Book,[37] which provides up-to-date information on side effects of vaccines, that noted GBS has been reported after hepatitis B vaccines, but "no causal association . . . has been demonstrated." Resp. Ex. C at 6.

Lastly, the WHO Information Sheet[38] for adverse reactions to the hepatitis B vaccine stated "there is no evidence of serious adverse events that have been causally linked to hepatitis B vaccination." Resp. Ex. C, Tab 2 at 1. With regard to GBS specifically, the WHO wrote that "on the basis of a careful review of all available evidence and advice from the Global Advisory Committee on Vaccine Safety (GACVS), WHO consider[ed] that the complete data do[es] not indicate a causal relationship between hepatitis B vaccine and GBS." Id. WHO acknowledged, however, that a "possible association between GBS and a receipt of the first dose of [the hepatitis B] vaccine was suggested" following the introduction of the plasma-derived[39] hepatitis B vaccine. Id. Additionally, "[a] review of case reports of adverse events and positive re-challenge of symptoms after hepatitis B vaccination has been interpreted as suggesting that vaccination could cause or trigger GBS in certain susceptible vaccine recipients." Id. (citing Pet. Ex. 19.2).

Dr. Leist also commented on Shaw et al. Resp. Ex. C at 5; see Pet. Ex. 18.3. He argued the current hepatitis B vaccine contains hepatitis B surface antigens that are distinct from earlier hepatitis B vaccines. Resp. Ex. C at 5. Thus, he asserted that the plasma-derived hepatitis B vaccine discussed in Shaw et al. is different that the vaccine at issue here.[40] Id. However, Dr. Leist did not explain how the recombinant vaccine would be less likely to trigger GBS.

---

[37] Respondent did not file a copy of the Pink Book.

[38] World Health Org. [WHO], Information Sheet: Observed Rate of Vaccine Reactions Hepatitis B Vaccine, (June 2012), https://www.who.int/publications/m/item/hep-b-vaccine-information-sheet.

[39] "Early vaccines were prepared by harvesting HBsAg from the plasma of people with chronic infection (plasma derived vaccine) while more recent ones are obtained by expressing plasmids containing the corresponding gene in yeast or mammalian cells (recombinant DNA vaccine)." Resp. Ex. C, Tab 2 at 1.

[40] There is no evidence in the record to show the specific vaccine Petitioner received.

Lastly, Dr. Leist agreed that peripheral neuropathies can be associated with hepatitis B infection, although he noted it is rare and "generally observed in individuals with chronic infection." Resp. Ex. C at 5. He opined that in such cases, "[i]t is thought to be due to immune-mediated neuronal damage secondary to the direct action of the virus itself on the nerve fibers or due [to] deposition of immune complexes of hepatitis B surface antigen the vasa nervorum, the small blood vessels that supply the peripheral nerves." Id. (citing Resp. Ex. C, Tab 1).[41] He added that "[v]iral replication also is an important factor contributing to disease activity and pathogenesis, as suggested by the high titers of hepatitis B virus DNA in such cases, and the improvement in clinical symptoms following implementation of antiviral therapy." Id.

In response to Dr. Kinsbourne's reliance on Yimam et al., which noted a temporal association between hepatitis B infection and GBS, Dr. Leist explained that the "[r]ecombinant hepatitis B vaccine does not contain [a] live virus that can replicate" and "[p]eripheral nerve involvement with hepatitis B virus generally depends on active replication of the virus." Resp. Ex. C at 5 (citing Pet. Ex. 18.5).

ii. Althen Prongs Two and Three[42]

Dr. Leist opined the hepatitis B vaccine at issue did not cause Petitioner's GBS. Resp. Ex. C at 7.

First, Dr. Leist noted Petitioner had no previous adverse reaction to any vaccine. Resp. Ex. C at 4. Because Petitioner tested positive for hepatitis B surface antibody and negative for hepatitis B core antibody and hepatitis B surface antigen in 2012, Dr. Leist opined that Petitioner had previously received one or more doses of the hepatitis B vaccine with no indication of an adverse event.[43] Id. Nor were there any reports of an adverse reaction following other vaccinations she received in 2013 and 2014.[44] Id. (citing Pet. Ex. 7 at 7, 25-28, 34). Additionally, following the administration of the hepatitis B vaccination at issue, Petitioner's

[41] Jeffrey A. Cohen et al., Mononeuropathy Multiplex Associated with Acute Hepatitis B Infection, 13 Muscle & Nerve 195 (1990).

[42] In Dr. Leist's recitation of the medical history, there are two errors. See Resp. Ex. C at 2-4. The first is a typographical error indicating Petitioner first presented to the ED at Putnam Hospital on March 4, 2015, but the correct date was May 4, 2015. Id. at 2; see Pet. Ex. 4 at 6. The second error is related to the lumbar puncture. He stated the lumbar puncture "was apparently not performed;" however, the procedure was attempted and unsuccessful. Resp. Ex. C at 3; see Pet. Ex. 5 at 162 (explaining that Dr. Pednekar was unable to collect CSF from the lumbar puncture on May 7, 2015).

[43] There is no record confirming any past receipt of a hepatitis B vaccination. Further, Dr. Leist did not explain why prior vaccination with hepatitis B influenced his opinions as to causation.

[44] Dr. Leist did not explain how this fact contributed to his opinion, if at all.

records did not indicate any immediate or delayed side effect in the hours and days following, nor any report of a local reaction at the injection site.[45] Id.

Next, he acknowledged that Petitioner's treating physicians noted a temporal association between the hepatitis B vaccine on April 23, 2015 and her GBS onset on May 3, 2015, but argued that none of Petitioner's treating physicians provided an opinion as to whether the vaccine was the cause of her GBS. Resp. Ex. C at 5, 7. For example, on May 6, 2015, ED physician Dr. Berkowitz noted Petitioner reported she "had a hep[atitis] [B] shot 1-1.5 [weeks] ago then [four] days ago started to exp[erience] bilat[eral] hand grip weakness and diff[iculty] walking [related to] weakness." Pet. Ex. 5 at 25. Dr. Berkowitz also stated that "[Petitioner] works in a health care facility and feels the symptoms may be related to the [h]epatitis B vaccine she received [one] month ago." Id. Neurologist Dr. Marks noted on May 7, 2015 that Petitioner had "[four] days of progressive weakness; [one] week after [h]ep[atitis] B vaccination." Id. at 96. Dr. Marks' discharge summary dated May 12, 2015 stated "[Petitioner] was given[] hep[atitis] B recently." Id. at 332. And Dr. Lasak, in September 2016, documented Petitioner "had [flu] A in April 2015, followed by [h]ep[atitis] B vaccination, and subsequent GBS." Pet. Ex. 7 at 118. Dr. Leist noted that while these physicians documented that Petitioner received the hepatitis B vaccine, none of them opined that it caused her GBS. Resp. Ex. C at 5.

Dr. Leist agreed the EMG performed on May 7, 2015 was consistent with GBS. Resp. Ex. C at 4-5. However, he noted "[a] serologic work-up to identify a possible cause[] of the [GBS] was not performed" and "[a] lumbar puncture was not performed."[46] Id. at 5.

Additionally, he noted Petitioner contacted her primary care office complaining of a breathing problem on April 9, 2015. Resp. Ex. C at 6 (citing Pet. Ex. 7 at 37). Dr. Leist acknowledged, however, that there are no records for care related to Petitioner having any breathing problem. Id. at 1, 6.

He also stated that Petitioner reported she had the flu in April 2015, citing a September 2016 record from Dr. Lasak for support. Resp. Ex. C at 6 (citing Pet. Ex. 7 at 118). But see Pet. Ex. 4 at 77 (noting Petitioner reported having the flu in February 2015); Pet. Ex. 4 at 223 ("[Petitioner] note[d] she also had a flu this winter."); Pet. Ex. 5 at 47 (documenting history of viral illness in February 2015). Dr. Leist conceded that there is no record from April 2015 indicating Petitioner had the flu. Resp. Ex. C at 1, 6.

Based on the records he cited above, Dr. Leist opined "it is possible that [Petitioner] had upper respiratory symptoms in the weeks before onset of symptoms of [GBS] on or about on May 3, 2015." Resp. Ex. C at 6. And, according to Yuki and Hartung,[47] "history of upper respiratory infectious symptoms or diarrhea [three] days to [six] weeks before the onset [of GBS]

[45] Again, Dr. Leist did not explain how this fact contributed to his opinion, if at all.

[46] See supra note 42.

[47] Nobuhiro Yuki & Hans-Peter Hartung, Guillain-Barré Syndrome, 366 New Eng. J. Med. 2294 (2012).

is not uncommon." Id. (quoting Resp. Ex. C, Tab 3 at 3). Dr. Leist explained, however, that his opinion "does not depend on whether or not she had such an infection." Id.

Regarding onset, Dr. Leist agreed Petitioner's GBS symptoms began on or about May 3, 2015. Resp. Ex. C at 6-7. He acknowledged there was a temporal association between vaccination and the onset of Petitioner's GBS. Id. at 7.

IV. DISCUSSION

A. Standards for Adjudication

The Vaccine Act was established to compensate vaccine-related injuries and deaths. § 10(a). "Congress designed the Vaccine Program to supplement the state law civil tort system as a simple, fair and expeditious means for compensating vaccine-related injured persons. The Program was established to award 'vaccine-injured persons quickly, easily, and with certainty and generosity.'" Rooks v. Sec'y of Health & Hum. Servs., 35 Fed. Cl. 1, 7 (1996) (quoting H.R. Rep. No. 908 at 3, reprinted in 1986 U.S.C.C.A.N. at 6287, 6344).

Petitioner's burden of proof is by a preponderance of the evidence. § 13(a)(1). The preponderance standard requires a petitioner to demonstrate that it is more likely than not that the vaccine at issue caused the injury. Moberly v. Sec'y of Health & Hum. Servs., 592 F.3d 1315, 1322 n.2 (Fed. Cir. 2010). Proof of medical certainty is not required. Bunting v. Sec'y of Health & Hum. Servs., 931 F.2d 867, 873 (Fed. Cir. 1991). Petitioner need not make a specific type of evidentiary showing, i.e., "epidemiologic studies, rechallenge, the presence of pathological markers or genetic predisposition, or general acceptance in the scientific or medical communities to establish a logical sequence of cause and effect." Capizzano v. Sec'y of Health & Hum. Servs., 440 F.3d 1317, 1325 (Fed. Cir. 2006). Instead, Petitioner may satisfy her burden by presenting circumstantial evidence and reliable medical opinions. Id. at 1325-26.

In particular, Petitioner must prove that the vaccine was "not only [the] but-for cause of the injury but also a substantial factor in bringing about the injury." Moberly, 592 F.3d at 1321 (quoting Shyface v. Sec'y of Health & Hum. Servs., 165 F.3d 1344, 1352-53 (Fed. Cir. 1999)); see also Pafford v. Sec'y of Health & Hum. Servs., 451 F.3d 1352, 1355 (Fed. Cir. 2006). The received vaccine, however, need not be the predominant cause of the injury. Shyface, 165 F.3d at 1351. A petitioner who satisfies this burden is entitled to compensation unless Respondent can prove, by a preponderance of the evidence, that the vaccinee's injury is "due to factors unrelated to the administration of the vaccine." § 13(a)(1)(B). However, if a petitioner fails to establish a prima facie case, the burden does not shift. Bradley v. Sec'y of Health & Hum. Servs., 991 F.2d 1570, 1575 (Fed. Cir. 1993).

"Regardless of whether the burden ever shifts to the [R]espondent, the special master may consider the evidence presented by the [R]espondent in determining whether the [P]etitioner has established a prima facie case." Flores v. Sec'y of Health & Hum. Servs., 115 Fed. Cl. 157, 162-63 (2014); see also Stone v. Sec'y of Health & Hum. Servs., 676 F.3d 1373, 1379 (Fed. Cir. 2012) ("[E]vidence of other possible sources of injury can be relevant not only to the 'factors unrelated' defense, but also to whether a prima facie showing has been made that the vaccine

was a substantial factor in causing the injury in question."); de Bazan v. Sec'y of Health & Hum. Servs., 539 F.3d 1347, 1353 (Fed. Cir. 2008) ("The government, like any defendant, is permitted to offer evidence to demonstrate the inadequacy of the [P]etitioner's evidence on a requisite element of the [P]etitioner's case-in-chief."); Pafford, 451 F.3d at 1358-59 ("[T]he presence of multiple potential causative agents makes it difficult to attribute 'but for' causation to the vaccination. . . . [T]he Special Master properly introduced the presence of the other unrelated contemporaneous events as just as likely to have been the triggering event as the vaccinations.").

B. Factual Issues

A petitioner must prove, by a preponderance of the evidence, the factual circumstances surrounding her claim. § 13(a)(1)(A). To resolve factual issues, the special master must weigh the evidence presented, which may include contemporaneous medical records and testimony. See Burns v. Sec'y of Health & Hum. Servs., 3 F.3d 415, 417 (Fed. Cir. 1993) (explaining that a special master must decide what weight to give evidence including oral testimony and contemporaneous medical records). Contemporaneous medical records, "in general, warrant consideration as trustworthy evidence." Cucuras v. Sec'y of Health & Hum. Servs., 993 F.2d 1525, 1528 (Fed. Cir. 1993). But see Kirby v. Sec'y of Health & Hum. Servs., 997 F.3d 1378, 1382 (Fed. Cir. 2021) (rejecting the presumption that "medical records are accurate and complete as to all the patient's physical conditions"); Shapiro v. Sec'y of Health & Hum. Servs., 101 Fed. Cl. 532, 538 (2011) ("[T]he absence of a reference to a condition or circumstance is much less significant than a reference which negates the existence of the condition or circumstance." (quoting Murphy v. Sec'y of Health & Hum. Servs., 23 Cl. Ct. 726, 733 (1991), aff'd per curiam, 968 F.2d 1226 (Fed. Cir. 1992))), recons. den'd after remand, 105 Fed. Cl. 353 (2012), aff'd mem., 503 F. App'x 952 (Fed. Cir. 2013).

There are situations in which compelling testimony may be more persuasive than written records, such as where records are deemed to be incomplete or inaccurate. Campbell v. Sec'y of Health & Hum. Servs., 69 Fed. Cl. 775, 779 (2006) ("[L]ike any norm based upon common sense and experience, this rule should not be treated as an absolute and must yield where the factual predicates for its application are weak or lacking."); Lowrie v. Sec'y of Health & Hum. Servs., No. 03-1585V, 2005 WL 6117475, at *19 (Fed. Cl. Spec. Mstr. Dec. 12, 2005) ("[W]ritten records which are, themselves, inconsistent, should be accorded less deference than those which are internally consistent." (quoting Murphy, 23 Cl. Ct. at 733 (1991)). Ultimately, a determination regarding a witness' credibility is needed when determining the weight that such testimony should be afforded. Andreu v. Sec'y of Health & Hum. Servs., 569 F.3d 1367, 1379 (Fed. Cir. 2009); Bradley, 991 F.2d at 1575.

Despite the weight afforded medical records, special masters are not bound rigidly by those records in determining onset of a petitioner's symptoms. Valenzuela v. Sec'y of Health & Hum. Servs., No. 90-1002V, 1991 WL 182241, at *3 (Fed. Cl. Spec. Mstr. Aug. 30, 1991); see also Eng v. Sec'y of Health & Hum. Servs., No. 90-1754V, 1994 WL 67704, at *3 (Fed. Cl. Spec. Mstr. Feb. 18, 1994) (Section 13(b)(2) "must be construed so as to give effect also to § 13(b)(1) which directs the special master or court to consider the medical records (reports, diagnosis, conclusions, medical judgment, test reports, etc.), but does not require the special master or court to be bound by them").

C. Causation

To receive compensation through the Program, Petitioner must prove either (1) that she suffered a "Table Injury"—i.e., an injury listed on the Vaccine Injury Table—corresponding to a vaccine that she received, or (2) that she suffered an injury that was actually caused by a vaccination. See §§ 11(c)(1), 13(a)(1)(A); Capizzano, 440 F.3d at 1319-20. Petitioner must show that the vaccine was "not only a but-for cause of the injury but also a substantial factor in bringing about the injury." Moberly, 592 F.3d at 1321 (quoting Shyface, 165 F.3d at 1352-53).

Because Petitioner does not allege she suffered a Table Injury, she must prove a vaccine she received caused her injury. To do so, Petitioner must establish, by preponderant evidence: "(1) a medical theory causally connecting the vaccination and the injury; (2) a logical sequence of cause and effect showing that the vaccination was the reason for the injury; and (3) a showing of a proximate temporal relationship between vaccination and injury." Althen, 418 F.3d at 1278.

The causation theory must relate to the injury alleged. Petitioner must provide a sound and reliable medical or scientific explanation that pertains specifically to this case, although the explanation need only be "legally probable, not medically or scientifically certain." Knudsen v. Sec'y of Health & Hum. Servs., 35 F.3d. 543, 548-49 (Fed. Cir. 1994). Petitioner cannot establish entitlement to compensation based solely on her assertions; rather, a vaccine claim must be supported either by medical records or by the opinion of a medical doctor. § 13(a)(1). In determining whether Petitioner is entitled to compensation, the special master shall consider all material in the record, including "any . . . conclusion, [or] medical judgment . . . which is contained in the record regarding . . . causation." § 13(b)(1)(A). The undersigned must weigh the submitted evidence and the testimony of the parties' proffered experts and rule in Petitioner's favor when the evidence weighs in her favor. See Moberly, 592 F.3d at 1325-26 ("Finders of fact are entitled—indeed, expected—to make determinations as to the reliability of the evidence presented to them and, if appropriate, as to the credibility of the persons presenting that evidence."); Althen, 418 F.3d at 1280 (noting that "close calls" are resolved in a petitioner's favor).

Testimony that merely expresses the possibility—not the probability—is insufficient, by itself, to substantiate a claim that such an injury occurred. See Waterman v. Sec'y of Health & Hum. Servs., 123 Fed. Cl. 564, 573-74 (2015) (denying Petitioner's motion for review and noting that a possible causal link was not sufficient to meet the preponderance standard). The Federal Circuit has made clear that the mere possibility of a link between a vaccination and a petitioner's injury is not sufficient to satisfy the preponderance standard. Moberly, 592 F.3d at 1322 (emphasizing that "proof of a 'plausible' or 'possible' causal link between the vaccine and the injury" does not equate to proof of causation by a preponderance of the evidence); Boatmon v. Sec'y of Health & Hum. Servs., 941 F.3d 1351, 1359-60 (Fed. Cir. 2019). While certainty is by no means required, a possible mechanism does not rise to the level of preponderance. Moberly, 592 F.3d at 1322; see also de Bazan, 539 F.3d at 1351.

V. ANALYSIS

A. Althen Prong One

Under Althen prong one, Petitioner must set forth a medical theory explaining how the received vaccine could have caused the sustained injury. Andreu, 569 F.3d at 1375; Pafford, 451 F.3d at 1355-56. Petitioner's theory of causation need not be medically or scientifically certain, but it must be informed by a "sound and reliable" medical or scientific explanation. Boatmon, 941 F.3d at 1359; see also Knudsen, 35 F.3d at 548; Veryzer v. Sec'y of Health & Hum. Servs., 98 Fed. Cl. 214, 223 (2011) (noting that special masters are bound by both § 13(b)(1) and Vaccine Rule 8(b)(1) to consider only evidence that is both "relevant" and "reliable"), aff'd, 475 F. App'x 765 (Fed. Cir. 2012). If Petitioner relies upon a medical opinion to support her theory, the basis for the opinion and the reliability of that basis must be considered in the determination of how much weight to afford the offered opinion. See Broekelschen v. Sec'y of Health & Hum. Servs., 618 F.3d 1339, 1347 (Fed. Cir. 2010) ("The special master's decision often times is based on the credibility of the experts and the relative persuasiveness of their competing theories."); Perreira v. Sec'y of Health & Hum. Servs., 33 F.3d 1375, 1377 n.6 (Fed. Cir. 1994) (stating that an "expert opinion is no better than the soundness of the reasons supporting it" (citing Fehrs v. United States, 620 F.2d 255, 265 (Ct. Cl. 1980))).

The undersigned finds Petitioner provided preponderant evidence that molecular mimicry is a sound and reliable theory to explain how the hepatitis B vaccination can cause GBS. There are several reasons for this finding, including expert opinions and medical literature.

The experts do not dispute the theory of molecular mimicry, or that it is a sound and reliable theory. Importantly, neither expert for Respondent refuted Petitioner's theory of molecular mimicry.[48] In fact, both experts for Respondent cited to the 2012 IOM report, which stated "molecular mimicry may contribute to the symptoms of GBS." Resp. Ex. A, Tab 2 at 2.

In support of his theory, Dr. Kinsbourne explained that after a vaccine is administered, the innate immune system detects epitopes of invading organisms and launches an immune attack on such epitopes. This process can lead to an attack against the structurally similar epitopes on an individual's own cells. GBS occurs when "those target self-epitopes are gangliosides on myelin sheaths and axons." Pet. Ex. 18 at 3.

Dr. Kinsbourne also cited three articles that discussed the homology between the hepatitis B virus and myelin basic protein. See Pet. Exs. 18.2, 19.1, 26. Specifically, "[t]he DNA sequence of [hepatitis B virus] was found to be homologous to myelin basic protein." Pet. Ex. 18.2 at 3; Pet. Ex. 19.1 at 2; Pet. Ex. 26 at 1.

[48] Dr. Leist did opine that in cases with hepatitis B infection, peripheral neuropathies, like GBS, are "thought to be due to immune-mediated neuronal damage secondary to the direct action of the virus itself on the nerve fibers or due [to] deposition of immune complexes of hepatitis B surface antigen the vasa nervorum, the small blood vessels that supply the peripheral nerves." Resp. Ex. C at 5. Although this theory is different than molecular mimicry, Dr. Leist did not opine that molecular mimicry, in the context of this case, was not sound or reliable.

Molecular mimicry has been accepted as a sound and reliable theory in many Vaccine Program cases dealing with demyelinating conditions, including GBS, forming the basis for petitioners to be entitled to compensation. See, e.g., Conte v. Sec'y of Health & Hum. Servs., No. 17-403V, 2020 WL 5743696, at *23 (Fed. Cl. Spec. Mstr. July 27, 2020) (noting the theory of molecular mimicry in a GBS case is "well-established and well-settled in the Vaccine Program"); Barone v. Sec'y of Health & Hum. Servs., No. 11-707V, 2014 WL 6834557, at *8-9 (Fed. Cl. Spec. Mstr. Nov. 12, 2014) (noting molecular mimicry "has been accepted in other Program cases as a reliable medical explanation for how various autoimmune conditions could develop after the receipt of different kinds of vaccinations"); Pierson v. Sec'y of Health & Hum. Servs., No. 17-1136V, 2022 WL 322836, at *23, *25-31 (Fed. Cl. Spec. Mstr. Jan. 19, 2022) (listing cases that have linked molecular mimicry to several demyelinating illnesses, including GBS, and various vaccines, including flu, tetanus-diphtheria-acellular pertussis, human papillomavirus, meningococcal, and hepatitis B, and finding it to be a sound and reliable theory in a pneumococcal conjugate/GBS case); J.G. v. Sec'y of Health & Hum. Servs., No. 20-664V, 2023 WL 2752634, at *30-32 (Fed. Cl. Spec. Mstr. Feb. 13, 2023) (finding molecular mimicry to be sound and reliable in a hepatitis A/GBS case).[49] Although decisions of other special masters are not binding, the undersigned finds these cases instructive and agrees with the reasoning of other special masters who have generally found molecular mimicry to be a sound and reliable mechanism for GBS. See Boatmon, 941 F.3d at 1358; Hanlon v. Sec'y of Health & Hum. Servs., 40 Fed. Cl. 625, 630 (1998), aff'd, 191 F.3d 1344 (Fed. Cir. 1999).

Moreover, in support of his theory, Dr. Kinsbourne cited studies examining the association between hepatitis B vaccination and GBS. Shaw et al. examined nine cases of GBS following hepatitis B vaccination. The authors found "[GBS] was reported significantly more often than expected," and using intervals of six and eight weeks, they found the incidence rate was "significantly higher" than the CDC background rate. Pet. Ex. 18.3 at 8. Although they concluded "no conclusive epidemiologic association could be made" between the hepatitis B vaccine and GBS, they noted GBS has been reported following many vaccines and infections, including the hepatitis B vaccine and infection. Id. at 1. Following the publication of Shaw et al., the CDC stated "surveillance of adverse reactions has shown a possible association between [GBS] and receipt of the first dose of plasma-derived hepatitis B vaccine." Pet. Ex. 12 at 11.

Souayah et al. examined VAERS reports of GBS following vaccination in 2004 and found 54 cases, and seven of the 54 were following vaccination with only hepatitis. Five of the 54 cases were following a combination of two or more vaccines that included a hepatitis vaccination. Although the authors did not specify which hepatitis vaccination was administered in these cases, they found the "[h]epatitis vaccine was the second most frequently associated vaccine with GBS." Pet. Ex. 18.4 at 2.

Dr. Kinsbourne also cited a number of case reports documenting GBS following hepatitis B vaccination. And each of these case reports discussed molecular mimicry as a mechanism by which the hepatitis B vaccine can cause GBS. Kakar and Sethi, for example, discussed the case

[49] The undersigned acknowledges these cases involve a different vaccine, although the same illness.

of a three-year-old child who developed GBS one day after hepatitis B vaccination. They noted molecular mimicry is a mechanism thought to cause demyelination and GBS because the DNA of hepatitis B virus was found to be homologous to myelin basic protein.

Khamaisi et al. discussed the case of a 52-year-old woman who developed GBS eight weeks after her second dose of a recombinant hepatitis B vaccine. Given the eight weeks between vaccination and onset, the authors found "[t]he temporal relationship . . . was suggestive of a vaccine-induced cause." Pet. Ex. 18.2 at 1. The authors also noted molecular mimicry has been suggested to trigger GBS, explaining that the "[h]epatitis B surface protein may provoke an autoimmune attack on a similar protein present in the nerve cells. In molecular mimicry involving T lymphocytes[,] these cells recognize their antigen as peptide-bound to MHC molecule." Id. at 3. Like Kakar and Sethi, Khamaisi et al. acknowledged "[t]he DNA sequence of [hepatitis B virus] was found to be homologous to myelin basic protein." Id.

Arhan et al. provided a case report of a 14-year-old male who developed GBS ten days after a hepatitis B vaccination. They explained that "[a]lthough the pathogenesis of hepatitis B vaccine associated GBS has not been completely elucidated," the mechanism of "[m]olecular mimicry between viral antigens and neural host tissues" has been proposed. Pet. Ex. 19.1 at 2. Similar to Kakar and Sethi and Khamaisi et al., Arhan et al. noted "[t]he DNA sequence of [hepatitis B virus] was found to be homologous to myelin basic protein." Id.

Vital et al. discussed two patients who received hepatitis B vaccinations. The first received a hepatitis B vaccine and developed symptoms consistent with ASAN 15 days thereafter. The second patient developed symptoms consistent with AMSAN 21 days after receipt of a hepatitis B vaccine. The authors opined the "autoimmune reaction was probably triggered by [hepatitis B vaccination]." Pet. Ex. 19.6 at 4.

Additionally, Dr. Kinsbourne cited a case report of GBS following infection with hepatitis B. Yimam et al. reported a case of GBS in a 42-year-old woman after hepatitis B infection. GBS "was thought to be secondary to acute [hepatitis B viral] infection." Pet. Ex. 18.5 at 2. Yimam et al. also noted infection with hepatitis B can cause GBS via molecular mimicry. The authors explained that "molecular mimicry between [hepatitis B virus] DNA and myelin basic protein, whereby initial host immunity to [hepatitis B virus] leads to the subsequent antibody-mediated attack of the myelin sheath." Id. at 3. The authors acknowledged that "[a]lthough the exact pathophysiology of GBS related to acute [hepatitis B virus] infection remains unclear, the association of these [two] conditions is well documented, as seen in this case report as well as earlier case reports." Id.

The undersigned is not persuaded by Respondent's experts' arguments. With regard to Dr. Moulton, the undersigned finds some of his opinions to be unsupported and speculative because they were based on assumptions that were not supported by literature or other foundational information. "[C]onclusory expert statements that are not themselves backed up with reliable scientific support" are consistently rejected. Kreizenbeck v. Sec'y of Health & Hum. Servs., No. 08-209V, 2018 WL 3679843, at *32 n.44 (Fed. Cl. Spec. Mstr. June 22, 2018), mot. for rev. denied, decision aff'd, 141 Fed. Cl. 138, aff'd, 945 F.3d 1362 (Fed. Cir. 2020). The undersigned does not rely on "opinion evidence that is connected to existing data only by the

ipse dixit of the expert." Prokopeas v. Sec'y of Health & Hum. Servs., No. 04-1717V, 2019 WL 2509626, at *19 (Fed. Cl. Spec. Mstr. May 24, 2019) (quoting Moberly, 592 F.3d at 1315). Instead, special masters are expected to carefully scrutinize the reliability of each expert report submitted. See id.

Additionally, a lack of supportive epidemiological evidence is not dispositive. First, "epidemiological studies cannot absolutely refute a causal connection" and "cannot prove a negative. It is always possible that another epidemiological study involving a bigger population will detect an increased risk not otherwise apparent in smaller studies." Harris v. Sec'y of Health & Hum. Servs., No. 10-322V, 2014 WL 3159377, at *11 (Fed. Cl. Spec. Mstr. June 10, 2014). Additionally, "[r]equiring epidemiologic studies . . . or general acceptance in the scientific or medical communities . . . impermissibly raises a claimant's burden under the Vaccine Act and hinders the system created by Congress, in which close calls regarding causation are resolved in favor of injured claimants." Andreu, 569 F.3d at 1378 (quoting Capizzano, 440 F.3d at 1325-26); see also Althen, 418 F.3d at 1280 (noting that "close calls" are resolved in a petitioner's favor).

The Federal Circuit has rejected arguments that rely heavily on statistics. See, e.g., Boatmon, 941 F.3d at 1363; Knudsen, 35 F.3d at 550 (rejecting the Respondent's theory because it was based on "[t]he bare statistical fact that there are more reported cases of viral encephalopathies than there are reported cases of [diphtheria-tetanus-pertussis] encephalopathies"); see also Hart v. Sec'y of Health & Hum. Servs., 60 Fed. Cl. 598, 605-10 (2004) (finding the special master's reliance on "bare" or "naked" statistics arbitrary and capricious and contrary to law). In Hart, the Court discussed statistical evidence and proof of causation and found "additional evidence adduced must show that the probabilities expressed are extendable to the facts of a given case and link the so validated statistical evidence into an otherwise plausible chain of causation." Hart, 60 Fed. Cl. at 609.

Dr. Moulton opines Petitioner did not provide "evidence bearing on causality []in the statistical or epidemiologic frameworks." Resp. Ex. A at 4. However, as explained above, such a requirement would impermissibly raise Petitioner's burden in this case. Thus, Petitioner has no obligation to provide statistical or epidemiological evidence. See, e.g., Capizzano, 440 F.3d at 1325-26 (explaining a petitioner need not make a specific type of evidential showing (i.e., epidemiologic studies) to satisfy her burden, and instead, may present circumstantial evidence and reliable medical opinions). Additionally, the undersigned does not find Dr. Moulton's opinions and arguments bearing on statistics, estimations, and assumptions persuasive based on case law where similar arguments have been rejected by the Federal Circuit. See, e.g., Boatmon, 941 F.3d at 1363; Knudsen, 35 F.3d at 550; see also Hart, 60 Fed. Cl. at 609. This is not the first time Dr. Moulton's opinions have been found less persuasive for these reasons. See Stevens v. Sec'y of Health & Hum. Servs., No. 99-594V, 2019 WL 2509626, at *21-22 (Fed. Cl. Spec. Mstr. Feb. 24, 2006).

Further, case reports alone may be insufficient to prove causation. However, where robust epidemiology studies are not available, they provide some evidence of causation. And here, where the medical literature reported GBS cases associated with hepatitis B infection and vaccination, the evidence weighs in favor of causation.

With regard to Dr. Leist's arguments, the undersigned finds many to be unsupported and underdeveloped. Dr. Leist appears to opine that it would be incorrect to rely on Shaw et al. because the hepatitis B vaccine in that study was plasma-derived, and not the current recombinant hepatitis B vaccine. However, he failed to provide any evidence in support of his argument. He did not explain the differences between the two vaccines, nor did he explain how the recombinant vaccine would be less likely to trigger GBS.

Considering the evidence as a whole, the undersigned finds Petitioner has proved a sound and reliable medical theory by preponderant evidence with respect to the first Althen prong.

B. Althen Prong Two

Under Althen prong two, Petitioner must prove by a preponderance of the evidence that there is a "logical sequence of cause and effect showing that the vaccination was the reason for the injury." Capizzano, 440 F.3d at 1324 (quoting Althen, 418 F.3d at 1278). "Petitioner must show that the vaccine was the 'but for' cause of the harm . . . or in other words, that the vaccine was the 'reason for the injury.'" Pafford, 451 F.3d at 1356 (internal citations omitted).

In evaluating whether this prong is satisfied, the opinions and views of the vaccinee's treating physicians are entitled to some weight. Andreu, 569 F.3d at 1367; Capizzano, 440 F.3d at 1326 ("[M]edical records and medical opinion testimony are favored in vaccine cases, as treating physicians are likely to be in the best position to determine whether a 'logical sequence of cause and effect show[s] that the vaccination was the reason for the injury.'" (quoting Althen, 418 F.3d at 1280)). Medical records are generally viewed as trustworthy evidence, since they are created contemporaneously with the treatment of the vaccinee. Cucuras, 993 F.2d at 1528. Petitioner need not make a specific type of evidentiary showing, i.e., "epidemiologic studies, rechallenge, the presence of pathological markers or genetic predisposition, or general acceptance in the scientific or medical communities to establish a logical sequence of cause and effect." Capizzano, 440 F.3d at 1325. Instead, Petitioner may satisfy her burden by presenting circumstantial evidence and reliable medical opinions. Id. at 1325-26.

The undersigned finds Petitioner has proved Althen prong two by preponderant evidence because Petitioner's clinical course was consistent with vaccine-related GBS and there is no persuasive evidence of an alternative cause.

First, the medical records support a clinical course consistent with the mechanism of molecular mimicry. Petitioner received a hepatitis B vaccine on April 23, 2015. On May 4, she presented to the ED complaining of hand and leg weakness for two days. She denied any other medical complaints or concerns. Two days later, on May 6, she returned to the ED due to increased weakness and an inability to walk. After various consultations and examinations, as well as an EMG on May 7, Petitioner was diagnosed with GBS and treated with five days of IVIG. All of Petitioner's treating physicians agreed Petitioner developed GBS. And the experts agree with the diagnosis.

Second, the undersigned finds Petitioner's treating physicians' statements provide some circumstantial evidence in support of Petitioner's claim. For example, on May 6, 2015, ED physician Dr. Berkowitz noted Petitioner reported she "had a hep[atitis] [B] shot 1-1.5 [weeks] ago then [four] days ago started to exp[erience] bilat[eral] hand grip weakness and diff[iculty] walking [related to] weakness." Pet. Ex. 5 at 25. Dr. Berkowitz added that "[Petitioner] works in a health care facility and feels the symptoms may be related to the [h]epatitis B vaccine she received [one] month ago." Id. Dr. Marks, a neurologist, noted on May 7, 2015 that Petitioner had "[four] days of progressive weakness; [one] week after [h]ep[atitis] B vaccination." Id. at 96. Dr. Marks' discharge summary on May 12, 2015 stated "[Petitioner] was given[] hep[atitis] B recently." Id. at 332. Dr. Lasak, in September 2016, documented Petitioner had "[h]ep[atitis] B vaccination, and subsequent GBS." Pet. Ex. 7 at 118.

The undersigned agrees with Dr. Leist that these statements from Petitioner's treating physicians do not amount to opinions on causation. However, these statements do note an association between vaccination and GBS. The undersigned finds that collectively, these statements constitute some circumstantial evidence that Petitioner's treating physicians associated her hepatitis B vaccine with the development of her GBS.

The undersigned does not find Dr. Leist's other opinions as to Althen prong two persuasive. First, he stated that Petitioner had no adverse reaction to any previous vaccination. However, he failed to explain how this fact contributed to his opinion that Petitioner's hepatitis B vaccine at issue here did not cause her GBS. Nor did he explain how a prior vaccination with hepatitis B influenced his opinion.

Additionally, the undersigned finds no persuasive evidence of an alternative cause. Dr. Leist asserts "it is possible that [Petitioner] had upper respiratory symptoms in the weeks before onset of symptoms of [GBS] on or about on May 3, 2015."[50] Resp. Ex. C at 6. However, the undersigned is not persuaded by this argument. The most contemporaneous-in-time records support a finding that Petitioner had the flu in February 2015, not April 2015 like Dr. Leist argues. If Petitioner did have the flu or a flu-like illness in February, it was several months prior to the onset of her GBS, and therefore too remote to have played a causal role.

Although there is documentation of a telephone call in which Petitioner complained of a "breathing problem" in April 2015, there is no visit for a "breathing problem" in or around April 2015. There is no medical record documenting any signs and symptoms of a cough, fever, sore throat, or other indices of an upper respiratory infection. Thus, the undersigned is not persuaded that Petitioner had an upper respiratory infection in the weeks prior to her GBS onset.

Furthermore, Dr. Leist expressed the opinion about Petitioner having an upper respiratory infection as a "possibility." Opinions that merely express the possibility—not the probability—are insufficient. See, e.g., Waterman, 123 Fed. Cl. at 573-74. And lastly, Dr. Leist noted that his opinion in this case "does not depend on whether or not she had such an infection." Resp. Ex. C at 6.

[50] The experts do not discuss Petitioner's UTI, diagnosed on May 4, 2015, as an alternative cause. Thus, the undersigned does not find that it was raised as a possible alternative cause.

For all of the reasons described above, the undersigned finds that Petitioner has provided preponderant evidence of a logical sequence of cause and effect required under Althen prong two.

C. Althen Prong Three

Althen prong three requires Petitioner to establish a "proximate temporal relationship" between the vaccination and the injury alleged. Althen, 418 F.3d at 1281. That term has been defined as a "medically acceptable temporal relationship." Id. Petitioner must offer "preponderant proof that the onset of symptoms occurred within a time frame for which, given the medical understanding of the disorder's etiology, it is medically acceptable to infer causation-in-fact." de Bazan, 539 F.3d at 1352. The explanation for what is a medically acceptable time frame must also coincide with the theory of how the relevant vaccine can cause the injury alleged (under Althen prong one). Id.; Koehn v. Sec'y of Health & Hum. Servs., 773 F.3d 1239, 1243 (Fed. Cir. 2014); Shapiro, 101 Fed. Cl. at 542; see Pafford, 451 F.3d at 1358. A temporal relationship between a vaccine and an injury, standing alone, does not constitute preponderant evidence of vaccine causation. See, e.g., Veryzer, 100 Fed. Cl. at 356 (explaining that "a temporal relationship alone will not demonstrate the requisite causal link and that [P]etitioner must posit a medical theory causally connecting the vaccine and injury").

Petitioner's expert, Dr. Kinsbourne opines Petitioner's GBS symptom onset was nine days after her hepatitis B vaccination, which is "within the risk interval for GBS" and "medically reasonable." Pet. Ex. 18 at 4. Respondent's expert, Dr. Leist, agrees Petitioner's GBS symptoms began on or about May 3, 2015, ten days post-hepatitis B vaccination. Resp. Ex. C at 6-7. He also acknowledges there was a temporal association. Id. at 7. The undersigned agrees that based on the medical records and expert opinions, Petitioner's symptom onset was on or about May 3, 2015, ten days post-hepatitis B vaccination.

To summarize, Petitioner received her hepatitis B vaccine on April 23, 2015. On May 4, 2015, Petitioner presented to the ED complaining of bilateral hand and leg weakness that began the day before, on May 3. Petitioner returned to the ED on May 6 reporting worsening weakness that began three days prior on Sunday, May 3. On May 6, "[Petitioner] needed assistance putting on her clothes and getting to the chair" and "came to the ED in a wheelchair." Pet. Ex. 4 at 87. GBS was listed as a differential diagnosis. Neurology consult with Dr. Sharf stated, "Good story for [GBS]." Id. at 78. Later that day, after transfer to the ED at Westchester Medical Center, Petitioner reported that "[four] days ago [she] started to exp[erience] bilat[eral] hand grip weakness and diff[iculty] walking [related to] weakness." Pet. Ex. 5 at 25. Neurologist consultation with Dr. Pednekar documented Petitioner's clinical course of bilateral weakness beginning Sunday (May 3, 2015). On May 7, Petitioner saw neurologist Dr. Marks, who documented Petitioner had "[four] days of progressive weakness." Id. at 96. Thus, Petitioner's contemporaneous medical records place onset on May 3, 2015, consistent with the opinions of Dr. Kinsbourne and Dr. Leist.

Additionally, the undersigned finds an onset of 10 days to be medically appropriate and supported by the expert opinions. First, this temporal association is consistent with the onset

period of three to 42 days as set forth in the Vaccine Injury Table for GBS following flu vaccination. 42 C.F.R. § 100.3(a)(XIV)(D).

Second, this timing is consistent with the articles and case reports cited by Petitioner. Shaw et al. examined nine cases of GBS following hepatitis B vaccination and all nine occurred within seven weeks of vaccination. Six of the seven patients who developed GBS following hepatitis vaccination in Souayah et al. had an onset within six weeks of vaccination. The patient in Kakar and Sethi developed GBS one day after hepatitis B vaccination. The patients in Vital et al. had onset periods of 15 and 21 days post-hepatitis B vaccination. Khamaisi et al. reported a case of GBS that occurred eight weeks following hepatitis B vaccination. Arhan et al.'s patient developed GBS 10 days after vaccination with hepatitis B. And the patient in Yimam et al. developed GBS within two weeks of hepatitis B infection.

Third, Respondent's expert, Dr. Leist, acknowledged the existence of a temporal relationship in this case. Resp. Ex. C at 7.

Lastly, this time frame has been acknowledged as appropriate in other Vaccine Program cases in which molecular mimicry has been proffered as the causal mechanism in a GBS case. See, e.g., Barone, 2014 WL 6834557, at *13 ("[S]pecial masters have never gone beyond a two-month (meaning eight week) interval in holding that a vaccination caused a demyelinating illness."); Pierson, 2022 WL 322836, at *32-33, *37 (finding a temporal relationship between vaccination and GBS when onset was within "the outermost medically appropriate onset date for vaccine-caused GBS at eight weeks, or 56 days, post-vaccination"); J.G., 2023 WL 2752634, at *34-35 (finding an onset of GBS 36 days post-hepatitis A vaccination to be medically appropriate).

Therefore, the undersigned finds the temporal association is appropriate given the mechanism of injury and Petitioner has satisfied the third Althen prong.

D. Alternative Causation

Because the undersigned concludes that Petitioner has established a prima facie case, Petitioner is entitled to compensation unless Respondent can put forth preponderant evidence "that [Petitioner's] injury was in fact caused by factors unrelated to the vaccine." Whitecotton v. Sec'y of Health & Hum. Servs., 17 F.3d 374, 376 (Fed. Cir. 1994), rev'd on other grounds sub nom., Shalala v. Whitecotton, 514 U.S. 268 (1995); see also Walther v. Sec'y of Health & Hum. Servs., 485 F.3d 1146, 1151 (Fed. Cir. 2007). As discussed above in the analysis related to Althen prong two, the undersigned found Respondent failed to establish evidence to show that Petitioner's GBS was caused by a source other than vaccination. Thus, Respondent did not prove by a preponderance of evidence that Petitioner's injury is "due to factors unrelated to the administration of the vaccine." § 13(a)(1)(B).

VI. CONCLUSION

For the reasons discussed above, the undersigned finds that Petitioner has established by preponderant evidence that her hepatitis B vaccine caused her GBS. Therefore, Petitioner is entitled to compensation. A separate damages order will issue.

IT IS SO ORDERED.

s/Nora Beth Dorsey
Nora Beth Dorsey
Special Master